DISSERTATION ON

SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER (14-20wks) IS A PREDICTOR OF GHT

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulation for the

award of the degree of

M.S. OBSTETRICS AND GYNAECOLOGY. BRANCH _II

THANJAVUR MEDICAL COLLEGE

THANJAVUR-613004

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI- 600032

APRIL-2016

CERTIFICATE

This is to certify that this dissertation entitled **"SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER IS PREDICTOR OF GHT"** is a bonafide original work of DR.V.MUNIRA in partial fulfillment of the requirements for M.S Branch II (obstetrics and gynaecology) examination to be held in APRIL 2016. The period of study was from September 2014 to August 2015.

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DECLARATION

I **Dr. V.MUNIRA**, solemnly declare that dissertation titled "**SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER IS PREDICTOR OF GHT**" is a bonafide work done by me at Thanjavur medical college, Thanjavur during September 2014-auguest 2015 under the guidance and supervision of **PROF.DR.E.KALARANI MD,DGO,** Head of the department of Obstetrics and Gynaecology, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to the Tamilnadu Dr.M.G.R Medical university towards partial fulfillment of the requirement for the award of M.S Degree (branch II) in obstetrics and gynaecology

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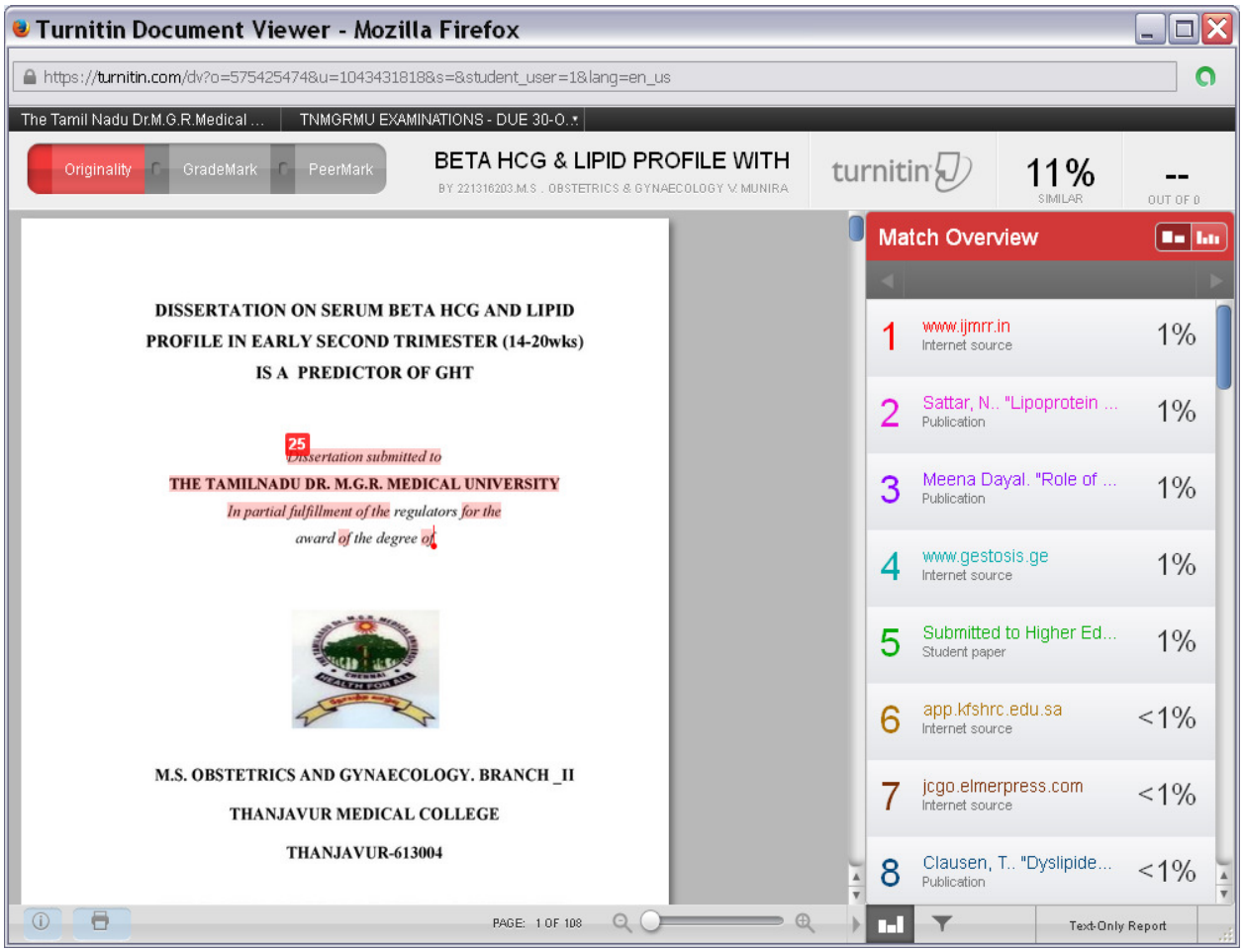
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This is to certify that The Research Proposal / Project titled

SERUM BETA HCG AND LIPID PROFILE IN EARLY SECOND TRIMESTER AS A PREDICTOR OF GESTATIONAL HYPERTENSION. (GHT)

submitted by Dr. V. MUNIRA. **of**

Dept. of OBSTETRICS AND GYNAECOLOGY **Thanjavur Medical College, Thanjavur**

was approved by the Ethical Committee.

Thanjavur

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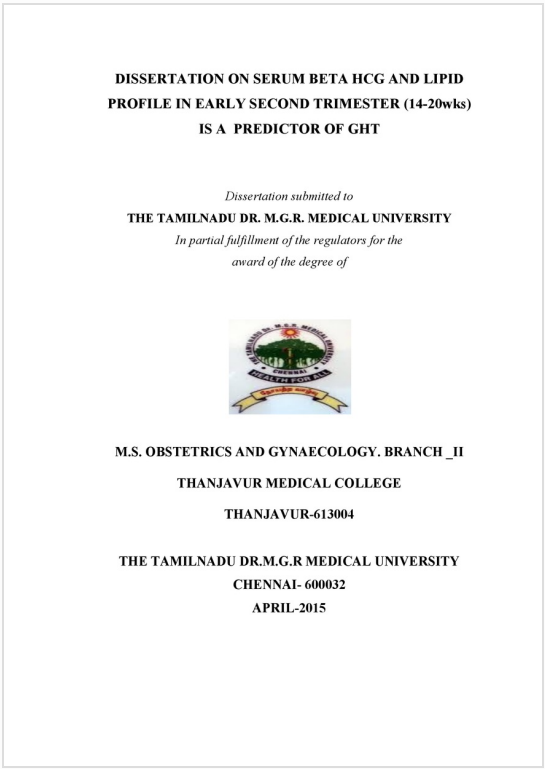

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CONTENTS

LIST OF FIGURES

LIST OF ABBREVIATIONS

Ach	Acetylcholine
ALT	Alanine amino transferase
AST	Aspartate amino transferase
BP	Blood pressure
B HCG	Beta human chorionic gonodatropins
CRP	C- reactive protein
CSF	Colony stimulating factor
DM	Diabetes mellitus
ECT	Extracellular fluid
EVT	Extra villous trophoblast
GHT	Gestational hypertension
GA	Gestational age
HELLP	Hemolysis, elevated liver enzyme and low platelets
HDL	High density lipoproteins
IGFI	Insulin like growth factor
IGF2R	Inslulin like growth factor receptor
IL	Interleukin

IL4R	Interleukin receptor
ISSHP	International society for the study of hypertension in pregnancy
IUGR	Intra uterine growth retardation
LDL	Low density lipoproteins
LDH	Lactate dehydrogenase
NK cells	Natural kill cells
NO	Nitrous oxide
PE	Pre eclampsia
PTD	Preterm delivery
ROS	Reactive oxygen species
RDS	Respiratory distress syndrome
SD	Standard deviation
SOD	Superoxide dismutase
THBSG	Thrombospondin 4
TNF	Tumour necrosis factor
TC	Total cholesterol
TG	Triglycerides
VLDL	Very low density lipoproteins
WHO	World health organisation

INTRODUCTION

Pregnancy is the most important period in women's life, but it can be dangerous also. Pregnancy is a physiological state with increased alteration in biochemical and mechanical processes if there are no pregnancy complications during pregnancy , the biochemical changes are reversible soon after delivery[1]. Hypertension and proteinuria are the important complications of pregnancy and are associated with high maternal and perinatal mortality and morbidity[2].

Gestational hypertension is defined as the presence of systolic blood pressure more than 140mm Hg and diastolic blood pressure more than 90mm Hg after 20 wks of gestation without proteinuria

Preeclampsia is defined as presence of systolic blood pressure above 140mmofhg and diastolic blood pressure above 90mmofhg, associated with proteinuria, edema (or) both.

If GHT mother develops generalised tonic clonic type of seizure is termed as eclampsia. It may occur before, during, (or) after delivery.

Sometimes these changes occur earlier when there is multi fetal pregnancy (or) extensive hydatiform changes in chorionic villi[3]

It usually occurs in 5- 10 percentage of pregnancies. It is specific to pregnancy and usually associated with high maternal and perinatal morbidity and mortality.

"BERG and COLLEGUES " reported that 16% 3201 of maternal death in the United States from 1991-1997 were complication of pregnancy induced hypertension. During this study black race had 3% higher mortality[4]. Preeclampsia in its severe variety is associated with cerebral (or) visual disturbances, oliguria, epigastric pain.

The elevation of blood pressure is the most important tool for identification of pregnancy induced hypertensive mother. Regular monitoring of blood pressure and urine albumin level is essential for diagnosing gestational hypertension.

The visual disturbances (or) oliguria, not a very common presentation, but can occur as a result of severe preeclampsia.

The patho physiological mechanism is characterised by a failure of the Trophoblastic invasion of spiral arteries, leading to maladaptation of the maternal spiral arterioles, so there is increased vascular resistance of the uterine artery and decreased perfusion of the placenta[5].

The spiral arterioles have a significant role in pre eclampsia. The structural and physiological changes in the spiral arterioles lead to the development of gestational hypertension.. Development of GHT is usually associated with change in serum beta HCG and lipid profile.

The peripheral edema in the GHT is due to the altered albumin level and decrease oncotic pressure leading to capillary leak throughout the body.

Vascular endothelial dysfunction also plays an important role in development of pregnancy induced hypertension, but pathophysiology remains unknown[6].

The results from many studies show the relationship between the aggravation of hypertensive complication and change in concentration of various chemical markers in mother's serum [7,8]. Interestingly change in serum beta HCG and lipid profile play an important role for early detection of GHT in early second trimester.

Most important features in GHT are supposed to be due to vasospastic phenomenon in kidney, uterus, placenta and brain. Altered lipid synthesis leading to decrease in PGI2 : TXA2 ratio is also supposed to be an important way of pathogenesis of GHT.

Abnormal lipid metabolism seems important in the pathogenesis of GHT too. Association of serum lipid prolife with GHT is highly suggested to reflect some new diagnostic tools. The hormonal imbalance is a prime factor for the etiopathogenesis of GHT and this endocrinal imbalance is well.

Measurement of serum lipid parameters may be of good predictive value in GHT avoiding the costly endocrinal investigations.

Maternal complications include:

1. Abruption placenta
2. Acute renal failure
3. HELLP syndrome
4. Eclampsia
5. Occipital lobe blindness
6. Pulmonary edema
7. Possible complication of caesarean section

Fetal complications include:

1. Prematurity
2. Intrauterine growth retardation
3. Intrauterine death
4. Fetal distress

These complications of gestational hypertension, could be prevented by prompt diagnosis , proper antenatal care and with timely intervention. When the GHT sets in very early trimester , close monitoring of the patient is necessary. If GHT women are on regular follow up and prompt medications, the risks are greatly reduced. Utero placental insufficiency is also reduced by controlling the blood pressure.

Decrease in platelet count may be associated with GHT which leads to severe bleeding tendencies. It may be the result of alteration in coagulation factors and fibrinogen. Regular monitoring of platelets and hepatic enzyme explains the needs for avoiding complication.

Termination of pregnancy is the treatment for severe preeclampsia/ eclampsia. The risk for both mother and fetus is more with continuation of pregnancy.

GHT may progress to severe preeclampsia/ eclampsia with uncontrolled hypertension. This is usually preventable by early detection of GHT using change in biochemical markers in maternal serum and prompt management of hypertension. Known GHT cases should be kept under regular follow up. They are advised to check blood pressure regularly, routine blood analysis and urine albumin monitoring. There are several methods to identify GHT which include isometric hand grip test, roll over test, angiotension II pressor response, mean arterial response test.

However these tests have some limitation for screening in view of false positive results and subjective nature of interpretation.

GHT complicates 5-10% of all pregnancies[9]. In the developed countries 16% maternal mortality is due to hypertensive disorders. Early detection of GHT and appropriate management is mandatory to decrease both maternal and perinatal morbidity and mortality.

REVIEW OF LITERATURE

1. Clausen et al[25] (1980) observed that women with triglycerides above 2.4mmol/l had increased risk (OR 5.1; 95% CI 1.1-23.1) of early onset pre eclampsia compared with those with triglycerides levels <1.5 mmol/l. He concluded that dyslipidemia before 20 weeks of gestation is associated with the risk of developing early but not late onset pre eclampsia.

2. Yamaguchi K et al[37] (1998) observed that women with triglycerides above 1.7mmol/l had increased risk (0R 6.0 96% RR) of development of pregnancy induced hypertension. He also concluded that women with elevated triglycerides (sensitivity 89% & positive predictive value 88%) are more prone to develop early onset pre eclampsia than the control population.

3. Vidayabati RK et al[24] & hijam et al[34] (2007) made a study and found that elevated beta HCG and lipid profile in early trimester are associated with development of GHT. They examined 164 pregnant women between 14-20 weeks of gestation. They found that there was a significant association with development of GHT with p <0.0001 valve.

In pregnant women with increased serum beta HCG has 10.7% increased chances of developing GHT. For one unit increase in total cholesterol there was 12.6% increased chance of developing GHT.

For every one unit increase in TG, VLDL, LDL there are 0.3% , 12.4% ,7.1% respectively increased chances of developing GHT. One unit increase in HDL has 11.4% less chance of developing GHT.

4. Evruke et al[41] (2004) observed that in gestational hypertensive patients only total lipid was different than the control group. (p=0.0003). In the chronic hypertension group , LDL, and total lipid levels were significantly different than the normotensive group. In the pre eclampsia group, total cholesterol (>2.5 MoM) , LDL cholesterol, total lipid and triglycerides were significantly different from the control group. He also concluded that there was no statistically significant change between trimesters for high density lipoprotein cholesterol.

5. Turpin et al[42] in the year 2003 made a study with lipid changes in pregnant women associated with the development of gestational hypertension. They concluded that women with elevated triglycerides level (> 3.0 MoM) had increased chances (17%) of developing GHT and early onset severe pre eclampsia. They also concluded there was a significant association between the elevated VLDL and development of GHT, with statistical significance (p <0.001).

6. Daniel A enqrobabrie, William et al[43], in the year 2003, investigated the relationship between the early pregnancy plasma lipid concentration and the risk of gestational hypertension. They identified that women who subsequently developed pre eclampsia had 10.4%, 13.6%, and 15.5% higher concentrations of LDL cholesterol, triglycerides and LDL/HDL ratios respectively with P value of < 0.05.The HDL cholesterol concentration were 7.0% lower in women with gestational hypertension than in the control groups with significant p value of < 0.05.

7. Gratacos et al[44] in the year 2001 made a prospective study and found that women with pre eclampsia had high levels of triglycerides (47% versus 13% in control subjects, p <0.001). They also concluded that women with elevated LDL level (56%) are more prone to have early onset pre eclampsia than the control group.

8. In 1995 Kaaja R, Trikkanen M.J, Ylikorkola O et al[42] explained that women with pre eclampsia shows,elevated level of lipoproteins in serum. For one unit increase in TG, VLDL, LDL there are 0.3%, 12.4%, 7.1% respectively increased chance of developing GHT. One unit increase in HDL women has 11.4% less chance of developing GHT.

9. Clause T, (1998) & Djurovic et al[25] (2001) found that dyslipidemia seems to be more efficient marker in predicting GHT at early second trimester with statistical significant (p <0.05).

Clause T did a study in 1998 in100 pregnant women; in that 54 % had elevated triglycerides & among them 48% developed GHT with statistical significant value.

10. Cekman et al[34] (2001) showed that plasma triglycerides and LDL levels were significantly higher in GHT subjects than controls. They did a study in 126 pregnant women, among which 78% had elevated triglycerides, out of which 56 people developed GHT with significant association ($P < 0.05$). In that study they identified the strength of association between LDL and GHT with an odds ratio 1 and relative risk of 3.2%.

11. Wakatsuki et al[36] (2000) & Williams et al[43] (2001) investigated the relationship between early pregnancy plasma lipid concentration and risk of GHT. They identified that women who subsequently developed GHT had 12.4% , 15.6% and 12.5% higher concentrations of LDL cholesterol, triglycerides and VLDL respectively with p value of <0.05. The HDL cholesterol concentration was 5.0% lower in women with GHT than in control groups with P value of < 0.05. They focused mainly on serum levels of LDL. They identified that there were significant relationship between elevated LDL & GHT. With this analysis they found that the sensitivity & positive predictive value of LDL against GHT was 86% & 92% respectively.

12. In the year 2003 Istam et al[36] identified that women with reduced level of HDL (less than 65 mg/dl) are more prone to develop GHT & early onset severe pre eclampsia. They did a study in 100 pregnant women. Women with reduced HDL level (42) are categorised as study group. They compared the HDL level in both the population & found that there was a significant correlation between GHT and reduced HDL level ($p<0.01$).

13. In the year 1996, Ray JG , Vermeulen MJ, Schull MJ et al[30] found that elevated levels of TG (>200 mg/dl) was associated with 4 times increased risk of development of GHT than normal. They found that strength of association was (RR) 4%. There is a significant correlation between TG & GHT with significant association.

14. In the year 1998 Lima VJ, Andrade CR, Ruschi et al[31] also found that TG contributes to the development of pre eclampsia, which is characterised by proteinuria and high blood pressure.

They analysed a total of 150 pregnant women, of which75 were GHT mothers &75 of them were normotensive. They did a TG analysis in both the groups. In GHT group 62 of them had elevated TG. In normotensive only 20 of them had elevated TG level. There was a high sensitivity and specificity between TG & GHT, 83.8% & 92% respectively.

15. In the year 1999 Yaron et al[21] found that elevated beta HCG was significantly associated with GHT. They did a study in 250 pregnant women with elevated beta HCG & found that beta HCG > 2.5 MoM was significantly associated with GHT.

16. In the year 1992 Aquilina & Ellis p et[23] al found a significant association between the beta HCG and elevated inhibin A associated with development of GHT and pre eclampsia. They did cross sectional study in 200 populations; in that women with elevated beta HCG more than 2.5 MoM in early second trimester were followed up till their delivery. 95 of them had elevated beta HCG value, of which 68 developed GHT. They found a correlation between beta HCG & GHT. They found that there was a statistical significance between beta HCG & GHT. They found that the sensitivity of beta HCG was 83% & specificity was 88%. Positive predictive value was 92%. With these findings they concluded that beta HCG can be used as a good predictor for GHT in early second trimester.

17 .In the year 2003 Vidyabati RK[25] , hijam danina et al[34] done a study with beta HCG and lipid profile in early second trimester. They examined 164 pregnant women between 14 -20 wks of gestation, in which twenty nine cases developed GHT, while 135 cases remained normotensive. The serum beta HCG increased very significantly ($p<0.001$) in those women who developed GHT. They proved that, for

one unit increase in the beta HCG, pregnant women had 3.7% increasing chance of GHT.

18. Wenstorm et al [45](2000) found that for every one unit increase in serum beta HCG, pregnant women had 10.7% increasing chances of developing GHT. For one unit increase in total cholesterol associated with 12.6% increased chance of developing GHT.

19. Pouta et al[41] found that increased level of serum beta HCG more than 25000 mIU /l is associated with the development of early onset severe pre eclampsia before 28 weeks of gestation & eclampsia. They did a study in 45 GHT women with elevated beta HCG value, in which 28 women developed severe pre eclampsia before 28 weeks of gestation. There was a statistical significant (p 0.01%) between elevated beta HCG & development of early onset severe pre eclampsia.

20. Morssink et al[43] found that cases with elevated beta HCG had increased incidence of GHT & early onset severe pre eclampsia. They found that one fold increase in beta HCG , had 12.4% increased chance of developing GHT. They concluded that, beta HCG has 92% sensitivity against GHT.

21. In 1999 taital stojikovic mikic research fellow found that elevated serum beta HCG in early second trimester is a useful predictor for early diagnosis of GHT. They published an American journal in the year 2000. A case control study was done in 250 women. Of them 125

were found to be normotensive & 125 were found to have GHT. Among the GHT population, 92 had elevated beta HCG. They did a statistical correlation between elevated beta HCG & GHT. With these results they concluded that women with elevated beta HCG had 7.2% increased chance of developing GHT.

22. In the year 2003 , Durick K, S krabin S, lesin jet et[28] al found that second trimester elevation of serum beta HCG is associated with development of GHT & low birth weight & IUGR babies. In the study they had 45 GHT women, of which 12 of them had low birth weight babies & 17 of them had IUGR babies & perinatal mortality was 14%.

23.Wenstrom KD quen J et al[45] (2000) found that there was an elevated second trimester serum beta HCG in singleton pregnancies and an adverse pregnancy outcome including development of GHT, IUGR, low birth weight babies and early onset severe pre eclampsia. In that study they had proven that most of the low birth weight babies were due to iatrogenic prematurity for severe pre eclampsia.

24. Gomen R, Perez R, David M et al (2003) identified that women with elevated serum beta HCG were associated with increased risk of development of GHT & IUGR. Women with elevated beta HCG more than 3.5 MoM were more prone to have IUGR babies than others. In this study they had high NICU admission (28%) in GHT population & most

of them got admitted for low birth weight. But there was no significant neonatal mortality.

25. In the year 2003Vallient et al[26] demonstrated that free beta HCG was a marker of high risk for GHT & associated with small for gestational age .They did a study in 192 pregnant women. Women with beta HCG > 3.5 MOM is taken as study group. In which 87 of them had elevated beta HCG. In the study group 24 of them developed GHT in third trimester. In which 12 of them had IUGR baby .They also found that women with elevated serum beta HCG were associated with increased risk of IUGR baby with statistical significance value ($p < 0.01$).

HYPERTENSIVE DISORDERS IN PREGNANCY

Hypertensive disorder complicating pregnancy is common and form one of the deadly triad that contributes for increased maternal mortality and morbidity

Identification of GHT in women should be done as early as possible. Treating GHT at the initial stage is easier when compared with treating patient with complications..Enhanced surveillance will be help in identifying high risk pregnant women .. Early detection and proper management helps to prevent progression of disease. Proper treatment at the initial stage of the disease is necessary for good pregnancy outcome for both mother and fetuS. However why pregnancy induces these vascular changes remains an unsolved problem in obstetrics.

CLASSIFICATION

According to National high blood pressure education program (2000)

Hypertensive disorder classified into 5 stages[9]

1. Gestational hypertension
2. Preeclampsia
3. Eclampsia
4. Preeclampsia super imposed on chronic hypertension
5. Chronic hypertension

GESTATIONAL HYPERTENSION

Defined as

1. Measurement of systolic blood pressure more than 140mmhg (or) diastolic blood pressure more than 90 mmofHg for the first time in pregnancy after 20weeks of gestation

2. Not associated with proteinuria

3. Blood pressure returns to normal within 12weeks of postpartum period.

PREECLAMPSIA

Defined as

1. Elevated blood pressure with proteinuria, edema may be present.

2. Proteinuria more than 300 mg in 24 hrs urine (or) 30 mg/dl. This is equal to 1(+) dipstick test in random urine sample.

Classified into two types:

1. Mild preeclampsia
2. Severe preeclampsia

Severe preeclampsia is considered if any of the following is present

- Systolic blood pressure more than 160 mmhg (or) diastolic blood pressure more than 110mmhg.
- Proteinuria 5gm per 24 hrs urine sample
- 2 (+) in dip stick random urine test
- Thrombocytopenia (platelets < 1laks/cumm)
- Symptoms like persistent headache, visual disturbance, epigastric pain
- Microangiopathic hemolysis
- Elevated serum creatinine
- Pulmonary oedema
- IUGR/ oligohydramnios
- Oliguria

ECLAMPSIA

If GHT mother develops generalised tonic clonic type of seizure is termed as eclampsia. It may occur before, during, (or) after delivery.

PREECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION.

Defined as known hypertensive patient developing proteinuria after 20 wks of gestation.

CHRONIC HYPERTENSION

Blood pressure more than 140/ 90 mmofHg before pregnancy (or) before 20 wks gestation (or) hypertension diagnosed after 20 wks of gestation and persistent after 12 wks of postpartum.

INCIDENCE & RISK FACTORS

GHT usually affects young and nulliparous women Incidence.(5-10%)

RISK FACTORS:

- Age (young & elderly primi)
- Interval from last pregnancy > 10 yrs
- Obesity BMI > 35kg/ m2
- Multifetal gestation
- Genetic predisposition
- Hydramnios
- Vesicular mole

- Hydrops fetalis
- Chronic hypertension
- Maternal diabetics
- Renal disease
- Anti phospholipids antibody syndrome
- Systemic lupus erythematosus
- H/O smoking
- Abnormal uterine artery Doppler at 18 -24 wks

AGE:

GHT is more common in young women. Incidence of preeclampsia & eclampsia is more in elderly women more than 35 yrs.

GENETIC FACTORS:

In 1873 Elliot described familial nature of the disease. This theory was reviewed by chesley (1968). He identified both parents contribute to genetic risk. There is presence of increased susceptibility of inherited genes from GHT mother to the fetus, the gene is capable of triggering GHT.

It is a "MULTI FACTORIAL POLYGENIC SYNDROME"[15]

Responsible genes:

1. MTHFR gene affecting methylene tetra hydrofolate reductase
2. Factor V (leiden) gene
3. Angiotensinogen gene (AGT)
4. HLA genes causing immunological tolerance
5. NOS3 gene affecting endothelial nitric oxide production
6. Prothrombin factor II gene
7. ACE gene

ETIOLOGY:

- Despite of lot of researches exact cause of GHT remains unknown
- More common in women who are
    1. Exposed to chorionic villi for the first time
    2. Exposed to superabundance of chronic villi (as with hydatiform mole/ twins)
    3. Women with pre-existing vascular disease
    4. Genetically predisposed

According to sibai (2003) the probable cause include

a. Abnormal Trophoblastic invasion of uterine vessels
b. Immunological intolerance between maternal & feto placental tissues

c. Maternal maladaptation to inflammatory /cardiovascular changes
d. Dietary deficiencies
e. Genetic influences

IMMUNOLOGICAL THEORY:

Immunological as well as genetic and endocrine mechanisms are involved in pathogenesis of GHT

The risk of GHT is increased with deficient blocking antibodies against the antigenic sites on the placenta (or) with increase no of antigenic sites on the placenta (Eg : multiple fetus). Some studies identified that there is no association between GHT & complement factor C3, C3F.

GENETIC THEORY:

In 1970 Cooper and Liston identified GHT is dependent upon single recessive gene but multi factorial inheritance cannot be excluded.

DIETARY DEFICIENCIES:

Calcium deficiency may be associated with GHT . Some studies revealed daily supplement of calcium 2g/day in mid trimester reduce the incidence of GHT

PATHOGENESIS

GHT women show changes in vasomotor activity, coagulation system, plasma volume , these disturbances may be due to endothelial cell dysfunction (or) activation

VASOSPASM :

Basic pathophysiology of GHT is " VASOSPASM"

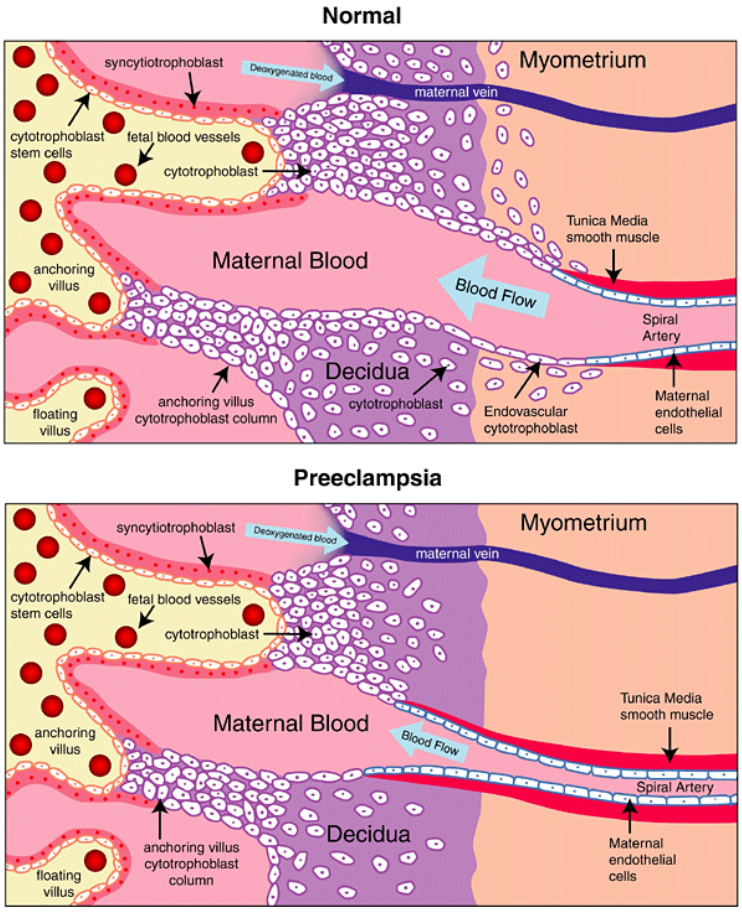
Vasoconstriction

Resistance to blood flow

Development of arterial hypertension

ABSENCE OF SPIRAL ARTERIOLES REMODELLING:

Pathogenesis of GHT starts with placenta.



DEVELOPMENT OF UTEROPLACENTAL VESSELS:

Occurs in two stages:

First stage of invasion:

This stage occurs upto 12 weeks. The cytotrophoblasts of the placenta invade the spiral arterioles upto the decidual-myometerial interface.

Second stage: (between 12-16 wks)

This stage involves invasion of intra myometrial segments of spiral arteries usually the cytotrophoblasts of the developing placenta migrates through the decidua and myometrium and invade the tunica media of the spiral arteries which supply blood to the fetus. These changes lead to transformation of small muscular arterioles to large low resistance vessels. These changes occur at end of the first trimester and completed by 18-20 wks..

In GHT spiral arteries fail to penetrate the myometrial segment. So spiral arteries remains narrow.. There is hypoperfusion of placenta leading on to placental ischemia.. Because of this there is a maternal endothelial dysfunction.

In 1980, Dewolf et al examined the arterioles from the placental implantation site using electron microscopy. They found that in early state there is damage of endothelial cells, accumulation of lipids in the myointimal cells resulting in narrowed lumen.

IMMUNOLOGICAL TOLERANCE:

- Immunologists studied that, abnormal immune protective mechanism, which is present in the mother against the fetus results in the development of GHT.
- To support this theory incidence of GHT is reduced in immunosuppressive patient.

- ❖ When compared with normotensive patient , GHT patient have reduced no of helper T cells in early second trimester.
- ❖ TH1/ TH2 imbalance was mediated by adenosine, which is found to be higher in GHT.

Placental changes in the GHT were similar with rejected kidney after transplantation. With this evidence, to support this theory, there is loss of maternal tolerance to the paternally derived placenta and fetal antigen. Women with GHT having reduced amount of messenger RNA for HLA –G. Cytokines mainly TNF alpha, IL 2 , IL 6 are the mediators of immune maladaptation in GHT patients.

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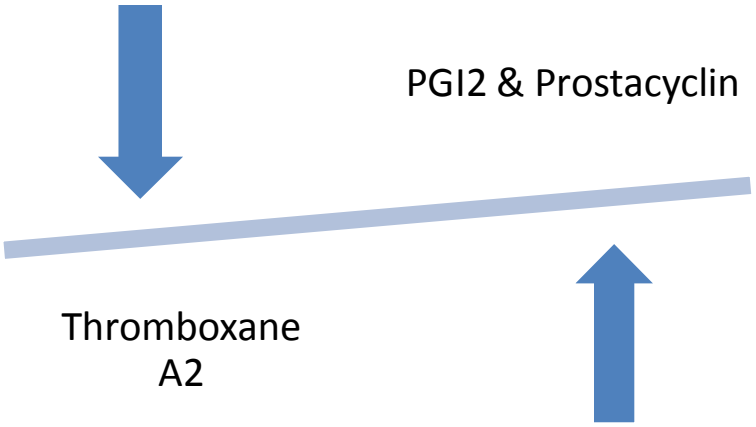
ENDOTHELIAL CELL ACTIVATION:

Intact endothelium has anti coagulant property. It releases the nitric oxide, so it reduces the vascular smooth muscle response to agonist. Damaged endothelium secretes pro coagulant, reduced nitric oxide production, so there is increased sensitivity to pressor agents.

PROSTAGLANDINS:



Prostacyclin : thromboxane ratio is decreased . Because of this decreased ratio, endothelial cells are more sensitive to vasopressor angiotensin II , which finally leads to vasoconstriction.

NITRIC OXIDE:

L- Arginine produces nitric oxide in endothelial cells. Nitric oxide is a potent vasodilator. In GHT patient NO production is reduced. Because of this there is increased sensitivity to vasopressor agents , ultimately there is increased mean arterial pressure.

OXIDATIVE STRESS:

This hypothesis explained that activated leukocytes are responsible for development of GHT . The decidua contains a large group

of cells, which on activation releases noxious agent like TNF alpha, leukotrienes. They are responsible for oxidative stress in the body.

ENDOTHELIN:

It is produced by human endometrium , it is a potent vaso constrictor. Endothelin levels are increased in GHT women. Some evidence suggests that magnesium sulphate decreases the endothelin 1 concentration.

ANGIOGENIC & ANTI ANGIOGENIC PROTEIN:

The balance between these proteins is responsible for normal development of placenta. In GHT there is increased production of anti angiogenic proteins, resulting in endothelial dysfunction. Use of anti angiogenic proteins in the prediction & diagnosis of GHT is a new research going on now.

2 Main anti angiogenic proteins used in research:

1. Soluble endoglin
2. Soluble FMS like tyrosine kinase 1

SOLUBLE ENDOGLIN:

It is a 65 kdalton , derived from placenta. It inhibits binding of TGF β binding with endothelial receptors which indirectly reduces nitric oxide release from the endothelium.

SOLUBLE FMS LIKE TYROSINE KINASE 1:

It reduces the vascular endothelial growth factor & placental endothelial growth factor and thus leads to endothelial dysfunction.

Both these anti angiogenic proteins are elevated in maternal serum before the development of GHT. So it can be used as an early marker for prediction of GHT.

RETENTION OF SODIUM:

In normal pregnancy plasma volume, glomerular filtration and renal flow increases, but in GHT all these parameters are reduced. This leads to retention of sodium which increases the vasopressor sensitivity.

All the signs & symptom of GHT & its complication can be explained by the response to generalised endothelial dysfunction.

1. Increased vascular permeability leads to proteinuria and edema
2. Changes in vascular tone of endothelial cells will cause hypertension
3. Procoagulants expression leads to coagulopathy

Endothelial dysfunction in the vasculature of brain, liver, kidney & placenta causes headache, seizure, epigastric pain, visual disturbances and intrauterine fetal growth restriction.

PATHOLOGICAL CHANGES:

GHT is a two stage disease

1. ASYMPTOMATIC STAGE:

Abnormal placental development during first trimester leads to placental insufficiency and release of placental materials in maternal circulation.

2. SYMPTOMATIC & PROGRESSION DISEASE:

Because of these substance, there was a development of hypertension, proteinuria, & organ involvement.

CARDIOVASCULAR SYSTEM:

Blood pressure = cardiac output X total peripheral resistance.

Normally total peripheral resistance decreases but in GHT it increases. This is the main reason for elevated blood pressure in GHT women. GHT women are more prone for pulmonary edema despite the normal ventricular function. This is due to endothelial – epithelial leak, compounded by decreased oncotic pressure from low albumin concentration.

HAEMATOLOGICAL SYSTEM:

Normally there is a disproportionate increase between plasma volume and RBC volume leading to physiological anemia of pregnancy. Plasma volume expansion is reduced in GHT . Intra vascular space is also contracted.

Because of this hemoconcentration hematocrit increases. Any attempt to expand the intra vascular space leads to increase in the pulmonary capillary wedge pressure which ends in pulmonary edema because of capillary leak.

Vasospasm is a main pathophysiology in GHT which leads to endothelial injury. Endothelial injury end up in micro angiopathic hemolysis, which results in anemia , fragmentation of RBC, thrombocytopenia.

BLOOD & COAGULATION:

The most common haematological abnormality is thrombocytopenia. It is more common in pre eclamptic patient which may be life threatening. Endothelial dysfunction causes release of tissue factor which act on coagulation system, resulting in disseminated intravascular coagulation.

THROMBOCYTOPENIA:

Described by stancke in 1922 in patients with severe GHT. All patients with GHT should be routinely examined for platelets. Intensity of thrombocytopenia depends on duration & severity of GHT. Less than 1 laks/cumm of platelet indicates severe disease. Thrombocytopenia is associated with higher maternal and fetal morbidity & mortality.

HELLP SYNDROME:

It is an acronym indicating

Hemolysis, Elevated Liver Enzyme, Low Platelets. It is common in severe preeclampsia which indicates hepatocellular necrosis.

COAGULATION:

1. Decreased anti thrombin III
2. Decreased protein C & S
3. Decreased plasma fibrinogen
4. Increased FDP
5. Increased fibrinopeptides A& B

ENDOCRINE SYSTEM:

Angiotensin II, catecholamine & vasopressin play an important role in elevation of blood pressure & increase the vascular resistance. Sensitivity of vascular endothelium to angiotensin II occurs 8-12 wks prior to the onset of clinical symptom.

Indomethacin & aspirin are prostaglandin inhibitors which decreases the angiotensin II sensitivity on the vascular endothelium.

RENAL SYSTEM:

In GHT there is increased resistance of renal afferent arterioles which leads to reduced renal perfusion. It also reduces the glomerular filtration rate. Glomerular endotheliosis also occurs in GHT which block the filtration barrier. Sodium concentration in urine is elevated.

RENIN ANGIOTENSIN ALDOSTERONE SYSTEM:

It is very important for maintainence of blood pressure & blood sodium level. In normal pregnancy plasma renin concentration& its activity, angiotensin II & aldosterone levels are elevated. Normal pregnant women have reduced refractoriness to angiotensin II.

But GHT patient lost their refractoriness to angiotension II. It is usually demonstrated by pressor response test at 18-22 wks of gestation.

LIVER :

Periportal haemorrhagic necrosis is the commonest pathological lesion in GHT. It usually manifest as upper epigastric pain & tenderness. It is associated with alanine transterase & aspartate transferase. It usually indicates severe form of disease. Area of infarct may be seen in liver which is usually diagnosed by CT/ MRI.

BRAIN:

Brain involvement in GHT was first diagnosed at autopsy specimen with severe disease. But now imaging studies give us very important information of GHT changes in the brain.

Common anatomical lesion:

1. Intracerebral haemorrhage
2. Cortical & sub cortical petechial haemorrhage
3. Haemorrhage in pons/ basal ganglia
4. Subcortical edema

Proposed theories behind the cerebral pathology

Theory : I

Due to vasospasm of cerebral vessels, there is diminished blood flow,to the brain resulting in ischemia, infaction, edema.

Theory: II

Sudden elevation of blood pressure may exceed the normal auto regulatory capacity of the cerebral vessels.

UTEROPLACENTAL PERFUSION:

Compromise in utero placental perfusion is common in GHT which is mainly due to vasospasm..

LIPID METABOLISM:

The hypothesized mechanisms for dyslipidemia and GHT association are as follows

1. Elevated plasma lipid and lipoprotein may induce endothelial dysfunction secondary to oxidative stress. They also noted that dyslipidemia may impair trophoblastic invasion thus contributing to a cascade of pathophysiological events that leads to development of GHT.
2. Pathological process of pre eclampsia via dysregulation of lipoprotein lipase resulting in a dyslipidemic lipid profile. Sera from pre eclamptic women had higher ratio of both FFA to albumin and increased uptake of FFA, which are further esterified to triglycerides.
3. Possible mechanism may be via metabolic syndrome. Metabolic characteristics of " insulin resistance syndrome" namely hyperinsulinemia and hyperuricaemia are also present in GHT. Women with history of pre eclampsia as compared with BMI matched counterparts without such a history, have higher circulating concentrations of fasting insulin, lipid and imflammatory coagulation factors years after delivery.

These genetic and environmental factors that contribute to the pathogenesis of metabolic syndrome and related vascular disorders may also be important in determining the occurrence of GHT

At present these hypothesis are the subjects of extensive investigations, for development of GHT

1. Placental ischemia
2. Altered endothelial cell function, possibly secondary to altered lipid metabolism
3. Immune maladaptation.

Serum triglyceride & total cholesterol were found to be high & HDL was significantly low in GHT patient.

Work in the field of cardiovascular research has shown that serum lipids have a direct effect on endothelial function and that abnormal serum lipid profiles are associated with endothelial dysfunction. As such the potential role of abnormal lipid metabolism in the genesis (or) expression of GHT is a subject of increasing interest. Lipids & lipoprotein levels undergo dramatic changes in pregnancy presumably to supply nutrients to the growing fetus. Plasma concentration of triglyceride & cholesterol increase approximately 30% and 50% respectively

- Although the presence of hyperlipidemia has been documented in a normal pregnancies the magnitude & significance of the lipid changes in pregnancies complicated by GHT has not been well defined. Increases in plasma cholesterol , triglycerides & VDRL has been noted

Histopathological finding noted in the uterine spiral arteries in patient with GHT may theoretically be associated with changes in serum lipid levels. In women with pre eclampsia , triglyceride concentrations climb substantially above these observed in normal pregnancy. It occur earlier before the appearance of clinical manifestations of the disorder.

- This study was undertaken in order to evaluate the lipid changes in pregnancies developing hypertensive disorders.

Total Plasma Lipid	: 400 – 600 mg/ dl
Total cholesterol	: 140 – 200 mg/ dl
HDL level	: 35-75 mg/dl
LDL level	: 80 – 130 mg/ dl
Triglycerides level	: 40-150 mg/dl
FFA levels	: 10- 20 mg/dl

One third of plasma lipids are cholesterol, one third is triglycerides & one third is phospholipid. Lipids are insoluble in water, they need the help of carriers in plasma . So they are complexed with proteins to form lipoproteins. The protein part of the lipoprotein is called apolipoprotein.

CLASSIFICATION OF LIPOPROTEINS:

There are five major classes of lipoproteins based on their separation by electrophoresis are

1. **CHYLOMICRONS:**

   They are synthesised in the intestine & transport exogenous triacylglycerol to various tissue. They consist of 90% quantity of lipid & 10% concentration of proteins. The chylomicrons are the least in density & longest in size.

2. **VLDL:**

   They are produced in liver and intestine and are responsible for the transport of endogenously synthesised triacylglycerols

3. **LDL:**

   Formed from VLDL, in the blood circulation. They transport cholesterol from liver to other tissues.

4. **HDL:**

Mostly synthesised in liver. They contain highest protein concentration. 3 different fractions of HDL (1, 2, 3). Identified by ultracentrifugation.It helps in transport of cholesterol from peripheral tissues to liver.

5. **FFA :**

Usually it is bound to albumin. Each molecule of albumin carry 20-30 molecules of free fatty acids. This lipoprotein cannot be separated by electrophoresis.

APOPROTEINS:

The protein part of lipoprotein is called apolipoproteins (or) apoprotein. All apoproteins are mainly systhesised in liver but small quantities are produced from almost all organs. Intestinal cells produce small quantities of apo- A. Apart from solubilising the lipid part, the protein components have specific functions. They are important in

1. Maintaining the structural integrity of the lipoprotein
2. Regulating certain enzymes which act on lipoproteins
3. Receptor recognition

METABOLISM OF LIPOPROTEINS:

Lipoprotein metabolism can be thought of as two cycles, one exogenous & one endogenous, both centered on the liver. These cycles are interconnected.

ENZYMES INVOLVED IN LIPID TRANSPORT:

1. Lecithin choseterol acyl transferase
2. Lipoprotein lipase
3. Hepatic lipase
4. Mobilizing lipase

DIAGNOSTIC IMPORTANCE OF LIPOPROTEINS:

The blood levels of certain lipoproteins have diagnostic importance. The ratio of HDL cholesterol to that in the LDL can be used to evaluate susceptibility to the development of heart disease. For healty person LDL/HDL ratio is 3: 5. Raised plasma LDL- Cholesterol concentration is associated with an increased risk of ischemic heart disease. Raised plasma concentration of HDL cholesterol is associatd with decreased risk of ischemic heart disease and seems to have protective effect.

Increased LDL and decreased HDL associated with increased risk of GHT. The exact nature of the protective effect of HDL is not known.

DISORDERS OF LIPOPROTEIN METABOLISM:

1. Hyperlipoproteinemia
2. Hypolipoproteinemia

HYPERLIPOPROTENEMIA:

Primary: familial hypercholesterolemia

Secondary :

1. Renal disease
2. Nephritic disease
3. Cirrhosis of liver
4. Hypothyroidism
5. Diabetes mellitus
6. Alcohol abuse
7. Women taking estrogen containing oral contraceptive.

(these disease are included in exclusion criteria)

HYPOPROTEINEMIA:

Primary: Abetalipoproteinemia

Tangier's disease

Secondary: severe malabsorption

Chronic liver disease

BETA HCG:

It is a glycoprotein with the highest carbohydrate content of any hormone in the human body. Like all glycoproteins , it consists of two subunits: α (92 amino acids) and β (145 amino acids), of which the α sub unit is structurally and immunologically similar in HCG ,LH,FSH and TSH.

Human chorionic gonadotropin is synthesised by the syncytiotrophoblast cells of the placenta even before implantation and can be detected in the peripheral blood by radioimmunoassay soon after implantation and well before the missed period.

The titres are increased in twin pregnancy, hydatidiform mole and choriocarcinoma. The titres are also high if the fetus has Down syndrome. The titres are low in ectopic pregnancy and threatened abortion.. The concentration of HCG is more on the maternal side and placenta than on the fetal side..

Placenta is the known primary trigger of pregnancy induced hypertension. Women with GHT usually have primary placentosis/ abnormal placentation. Beta HCG is secreted in abundance from placenta. Beta HCG in mid trimester is elevated in patients with chromosomally normal fetus who later on develop pre eclampsia.

There is a general agreement that the placenta remains the main source of beta HCG in patients with GHT, but whether the cause of high circulating levels of hormone is placenta overproduction is still debated. It is may be due to abnormal placental invasion (or) placental immaturity.

It may also be linked to the trophoblast response to hypoxia with the development of a hypersecretory state. A role of placental factors is further supported by findings of increased peroxidation and oxidation stress in placentas of women with GHT. Oxidative stress secondary to reduced placental perfusion leads to endothelial dysfunction linking the two stages of the syndrome.

- Compared with normal pregnancies the placenta of patients with unexplained elevated maternal betaHCG levels in the seond trimester tend to be larger and tend to have an increased density of betaHCG positive trophoblast along with an increased intensity of betaHCG immunostaining within the placental villi

NORMAL HCG LEVEL DURING PRGNANCY:

Human chorionic gonadotropin can be detected in maternal blood and urine 8-10 days after fertilization . The serum level is 100 mIU/ml at 2 weeks after fertilization, peaks to 100,000 mIU/ml at 10 weeks and declines. By 14 weeks , a nadir is reached (20,000- 25,000) and maintained for the rest of the pregnancy.

Estimation of serum β HCG levels was done by enzyme linked immune absorbent assay. The quantitative determination of chorionic gonadotropin in serum carried out by microplate immune enzymometric assay, with kits obtained from monobind.

HIGH LEVELS OF VALUE:

Seen in Multiple pregnancy , gestational Trophoblastic disease, Down syndrome, erythoblastosis fetalis.

LOW LEVELS OF VALUE:

Found in ectopic pregnancy and early pregnancy loss. Both these category patients were excluded from the study.

COMPLICATION:

MATERNAL:

CENTRAL NERVOUS SYSTEM:

- Eclampsia
- Cerebrovascular accident
- Retinal detachment
- Cortical blindness

RESPIRATORY SYSTEM:

- Pulmonary edema
- Acute respiratory distress syndrome

RENAL SYSTEM:

- Renal cortical necrosis
- Renal tubular necrosis
- Renal failure

LIVER :

- HELLP syndrome
- Hepatic rupture : most serious complication
- Fatty liver: very rare disease characterised by microvesicular hepatic steatosis

HAEMATOLOGICAL :

1. Disseminated intra vascular coagulation

PLACENTA:

- Abruption placenta

FETAL COMPLICATION:

"GHT is considered as maternal disease, fetus is incidental participant, in fetal point of view it is a fetal disorder, and mother is incidental participant".

Normal placental changes which is more extensive in case of pre eclampsia includes

1. Increased syncytial knots
2. Increased number of true infarcts
3. Cytotrophoblast proliferation
4. Villous necrosis
5. Maternal decidual arteries fibrinoid degeneration.

Because of this there is

1. Increased secretion of chorionic gonodotropin
2. Increased secretion of steroid hormones
3. Deterioration of acute transport mechanism of vital aminoacids

All the factors lead to: 1. Fetal hypoxia and IUGR

2. IUD
3. Prematurity- mainly iatrogenic
4. Respiratory distress syndrome
5. Drug induced side effects in fetus

HELLP SYNDROME:

HELLP syndrome was first discovered by Weinstein, in the year 1982.

It comprises Hemolysis, Elevated liver enzyme, Low platelet count. Incidence is about 0.2- 0.6%. It usually present with nausea, vomiting, epigastric pain.

CRITERIA FOR DIAGNOSIS:

HEMOLYSIS:

1. Abnormal peripheral smear: burr cells, schistocytes
2. Bilirubin: > 1.2 mg/ dl
3. Lactate dehydrogenase > 600 u/l
4. Low serum haptaglobulin

ELEVATED LIVER ENZYMES:

1. SGOT > 70 u/l
2. LDH > 600 u/l

LOW PLATELETS: platelet count < 1 laks/cumm

DIFFERENTIAL DIAGNOSIS:

- ✓ Cholestasis of pregnancy
- ✓ Acute fatty liver of pregnancy
- ✓ Viral hepatitis
- ✓ Haemolytic uremic syndrome
- ✓ Thrombotic thrombocytopenic purpura

MANAGEMENT:

- ✓ Termination of pregnancy
- ✓ Prophylactic anti- convulsant to be given
- ✓ Platelet count and LDH level return to normal within 72 hrs after delivery. So it can used as markers for follow up.

PREDICTION AND PREVENTION OF GHT:

Several attempts were done to find out the early markers of impaired placenta perfusion, faulty placentation, & endothelial cell activation and dysfunction. Till now there is no reliable test to predict pre eclampsia.

Conde agudeolo and associates , provided the review for the tests in the year 2009.

Tests for vascular resistance:

1. Isometric handgrip/ cold pressor test
2. Angiotensin II infusion test
3. Mid trimester mean arterial pressure
4. Platelet angiotensin II binding
5. 24 hours ambulatory blood pressure monitoring
6. Uterine artery Doppler
7. Fetal transcranial Doppler velocimetry

Tests for placental unit endocrine dysfunction:

1. Human chorionic gonadotropin
2. Alpha feto protein
3. Estriol
4. Pregnancy associated protein A
5. Inhibin A
6. Activin A
7. Placental protein 13
8. Corticotrophin releasing hormone

Test for renal dysfunction :

1. Serum uric acid
2. Micro albuminuria
3. Urinary calcium/ kallikrein
4. Microtransferrinuria
5. N – acetyl –β- glucosaminidase

Test for endothelial dysfunction:

1. Platelet count
2. Fibronectin
3. Endothelial adhesion molecule
4. Prostaglandins and thromboxane

5. C- reactive protein and cytokines

6. endoglin

Miscellaneous:

1. Anti thrombin III
2. β 2 glycoprotein
3. Atrial natriuretic peptides
4. lipid profile

Mean arterial blood pressure:

MAP = diastolic pressure + 1/3 pulse pressure

Usually there is a normal fall in blood pressure in mid trimester. Those who don't have normal fall with elevated mean arterial pressure more than 90 mm hg in second trimester are at risk of developing pre eclampsia.

Roll over test:

Ask the women to turn from left lateral to supine position. If increase in diastolic blood pressure 20 mmof Hg (or) more is considered as test positive.

Angiotensin sensitivity test:

In normal patient there is refractoriness to angiotensin between 28 – 32 wks of gestation, it is usually lost in GHT. This test is based on above fact infuse angiotensin < 8 ng/kg/min, if the pressor response occurs with the above dose they are more likely to develop GHT. Sensitivity is about 80% but it is invasive test.

Uterine artery Doppler:

Non pregnant women:

Reduced diastolic flow and notching of uterine artery

Normal pregnancy:

Because of Trophoblastic invasion, flow increased and notch disappears.

"PERSISTENCE OF DIASTOLIC NOTCH (OR) INCREASED RESISTANCE IN THE UTERINE ARTERY AT 20 -22 Wks indicate DEFECTIVE TROPHOBLAST INVASION".

The latest combination method for prediction of GHT is uterine artery Doppler with measurement of PAPPA – A & PLACENTAL PROTEIN 13.

METHODS OF PREVENTION:

1. Low Salt Diet:

Earliest research found that salt restriction will prevent GHT (De Snoo 1937). Knuist & collegues reported in 1998 , salt restriction not preventive for GHT.

2. Calcium Supplementation:

Calcium supplementation with 1.5-2g calcium carbonate (or) elemental calcium from various preparations has shown to reduce the incidence of pre eclampsia almost by the half. The beneficial effect was greatest for high risk women, and those with low calcium intake.

3. Fish Oil Supplementation:

In 2006 makrides & olatsdottir & collegues and oslin (2000) fond that there was no benefit with fish oil supplementation.

4. Anti – Oxidiants:

Naturally occurring anti oxidants are vitamin C & E. Reduction in these anti- oxidants are associated with increased risk of GHT (rajimaters & associates 2004). Thus supplementation of anti – oxidants improves the oxidative property of women , who has increase risk of GHT. But studies (poston 2006, rumbold 2006) revealed that this anti- oxidants will not reduce the risk of pre eclampsia.

5. **Anti Thrombotic Agent:**

6. **Low Dose Aspirin:**

In 1986 wallenburg and associates revealed that daily oral dosage of 50 -150 mg of aspirin inhibits thromboxane A2 synthesis, it reduce the relative risk of GHT by 10%.

7. **Low Dose Aspirin & Heparin:**

High incidence of thrombotic lesion in placenta was found in GHT patient. In 2006 sergis and associates found that low dose aspirin with low molecular heparin improve the pregnancy outcome in GHT patient when its compare with low dose aspirin alone.

OBJECTIVES

1. To test the hypothesis that women with high serum beta HCG & lipid profile in early second trimester have risk of developing hypertensive disorder of pregnancy
2. To identify the "at risk" women earlier may help in taking timely prevention and curative management and to prevent complications associated with GHT.
3. To study the strength of association between the elevated beta HCG & lipid profile with GHT

MATERIALS AND METHODS

This was a prospective study, conducted in OP population at Raja Mirasudar Hospital, Thanjavur from Sep 2014- aug 2015. Total no of 100 pregnant women who attended antenatal clinic of department of O&G at RMH Thanjavur were included. All the patient were screened for serum beta HCG & lipid profile in early second trimester between (14-20 wks) ,to be followed up till their delivery. The study groups involved both primi & multi gravida. They were selected on the basis of simple random sampling.

Comparative study of serum beta HCG and lipid profile performed between those who remain normotensive and those who develop GHT. GHT was defined as systolic blood pressure more than 140mmmhg and diastolic blood pressure more than 90mmhg occurring on two (or) more occasions after 20 wks of gestation recorded 6 hours apart. Preeclampsia is defined as gestational hypertension and proteinuria of at least (2+) / 1g/dl on dip stick (or) 24 hrs urinary protein excretion > 0.3 g.

Fasting venous blood sample (3ml) was collected and tests were carried out on the same day. Estimation of serum beta HCG level was done by enzyme linked immune absorbant assay. The quantitative

determination of chorionic gonodotropin in human serum was carried out by micro immune enzymatic assay with kits obtained from monokind Inc osta mosa.

Serum lipid profile estimation was done by enzymatic calorimetric test with lipid clearing factor (LCF) using kits. LDL cholesterol & VLDL cholesterol valves in mg/dl are indirectly measured . The cases were followed up regularly in the antenatal OPD till delivery. All the detailed data were collected from the delivery log book. Data was analysed statistically. The study group participated voluntarily in this study and each of them gave an informed consent. This study was approved by ethical and research committee of Thanjavur medical college.

INCLUSION CRITERIA:

1. Patient with previous history of GHT
2. AN mother in gestational age of (14-20 wks)

EXCLUSION CRITERIA:

1. Hypertension diagnosed before 20 wks of gestation
2. Diabetics
3. Multiple pregnancy
4. Molar pregnancy

5. Hypothyroidism
6. USG proven fetal congenital malformation
7. H/O hypercholesterolemia

CUT OFF VALVES

	NORMAL	**ABNORMAL**
Beta HCGmIU/l	20000-25000	>250000
TG mg/dl	< 200	>200
VLDL mg/dl	< 40	>40
TC mg/dl	<200	>200
LDL mg/dl	< 130	>130
HDL mg/dl	>65	<65
BP mmofHg	110-138/72-88	140/90

METHOD OF COLLECTION OF SAMPLE:

Samples were drawn after an overnight fasting from the antecubital vein 3 ml whole blood was collected in red capped vaccum tube containing clot activator and left at room temperature to clot for half an hour. Serum was separated by centrifugation at 1200rpm and analysed using Siemens dimension .

RMH - OPD

DEPARTMENT OF BIOCHEMISTRY

METHODOLOGY OF LIPID PARAMETERS:

Parameters	Methodology
Total cholesterol	CE/CO/HPO
HDL cholesterol	Accelerator selective detergent
LDL cholesterol	Detergent / CE/ CO/POD
Triglycerides	LPL/GK/GPO/POD

ESTIMATION OF TOTAL CHOLESTEROL:

Principle:

Cholesterol esterase catalysethe hydrolysis of cholesterol esters to produce free cholesterol which, along with pre existing free cholesterol oxidase to form cholest 4-ene-3one and hydrogen peroxide. In the presence of hydrogen peroxidase (HPO).. The hydrogen peroxide thus formed is used to oxidize N, N diethyl aniline- HCL / 4 amino triptyline is directly proportional to the total cholesterol concentration and is measured using a polychromatic (453,540,700 nm) end point technique.

$$\text{Chosterol estors} \xrightarrow{\text{Cholesterol esterase}} \text{cholesterol + fatty acid}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{cholest 4-ene-3one} + H_2O_2$$

$2H_2O_2$ +DEA.HCL/AAP----------4 H_2O + oxidized DEA. HCL/AAP

Potassium oxalate/ sodium fluoride can decrease cholesterol results an average of 12%. li heparin can depress cholesterol results by an average of 4gm/ dl t a level of 200 mg/ dl

ESTIMATION OF HDL CHOLESTEROL:

Principle:

The HDL cholesterol assay is homogenous method for directly measuring HDL – C levels without the need for off line pre treatment (or) centrifugation steps

Accelerator selective detergent methodology

HDL,LDL,VLDL,Chylomicrons-------- LDL,VLDL,chylomicrons.

HDL ---------- HDL disrupted

HDL cholesterol ---------- cholesterone + H_2O_2

H_2O_2 + DSBM+4-AAP ---------- colour department

(Wavelength 600nm & 700nm)

ESTIMATION OF LDL CHOLESTEROL:

Nonsoluble LDL-C, VDRL –C ---------- soluble non- LDL- C

Soluble non-LDL-C ----------non colour forming

Non soluble LDL-C ------- soluble LDL-C

Soluble LDL + O_2----------cholestenone + H_2O_2

H_2O_2+ DSBMT+ 4-AA---------- colour development

ESTIMATION OF TRIGLYCERIDES:

Triglycerides---------- glycerol+ fattyacids

Glycerol+ ATP ----------glycerol -3- phosphate + ADP

Glycerol-3-phosphate+O_2 ----- dihydroxyacetone phosphate Po_4

$2H_2O_2$+aminoantipyline----------quinoneimine+ HCL+ 4 H_2O

(wavelength 510nm & 700 nm)

BETA HCG:

The HCG quantitative test is also known as quantitative serial beta HCG, Repeat quantitative beta HCG, beta HCG blood test and quantitative blood pregnancy test.

Quantitative measurement of beta HCG was done by ELISA technique. Blood sample were collected with all aseptic precautions. Serum was separated by centrifugation. All reagents were brought to room temperature. 25 µL of serum samples were put into microplate well. 100µL of enzyme reagent (β HCG) were added to all wells, incubated and contents were discarded.100 µ L of working substrate solution was added and incubated. Then 50µ L of stop solution was added and absorbance in each well was read by ELISA reader.

STATISTICAL ANALYSIS

Data was expressed in terms of mean +/- SD. Chi square test was applied to estimate the difference between positive groups. Unpaired 't' test was used to study the changes in the beta HCG and lipid profile values.

P value > 0.05 was taken as non significant

P value < 0.05 was taken as significant

P value < 0.01 was taken as highly significant

P value < 0.001 was taken as very highly significant

RESULTS

The results obtained from this present study were categorised according to their initial beta HCG & lipid profile level.

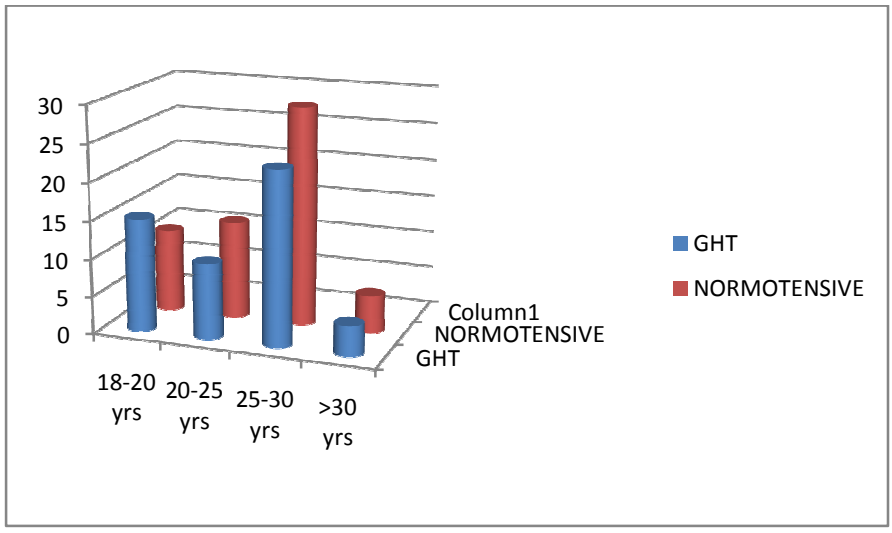
Those who had elevated beta HCG & LIPID PROFILES categorised in study group.

Following parameters are analysed:

1. Serum β HCG
2. TG
3. HDL
4. VLDL
5. LDL

TABLE :1 AGE GROUP (years)

GROUPS	18-20	21-25	26-30	>30
GHT	5	10	23	4
NORMOTENSIVE	11	13	29	5
TOTAL	26	23	52	9



The age group of the subject was between 18-30 years. The mean age and standard deviation in GHT was 24.48 ± 2.08 years and in normotensive it was 23.65±2.47 years. There was no statistical difference.

TABLE 2: GRAVIDA

GROUPS	PRIMI	G2	G3	G4
NEGATIVE	15	13	13	4
Beta HCG (+) Ve	5	2	12	1
Lipid profile (+) Ve	4	13	1	0
Both (+)Ve	15	11	10	1
Total	39	29	26	6

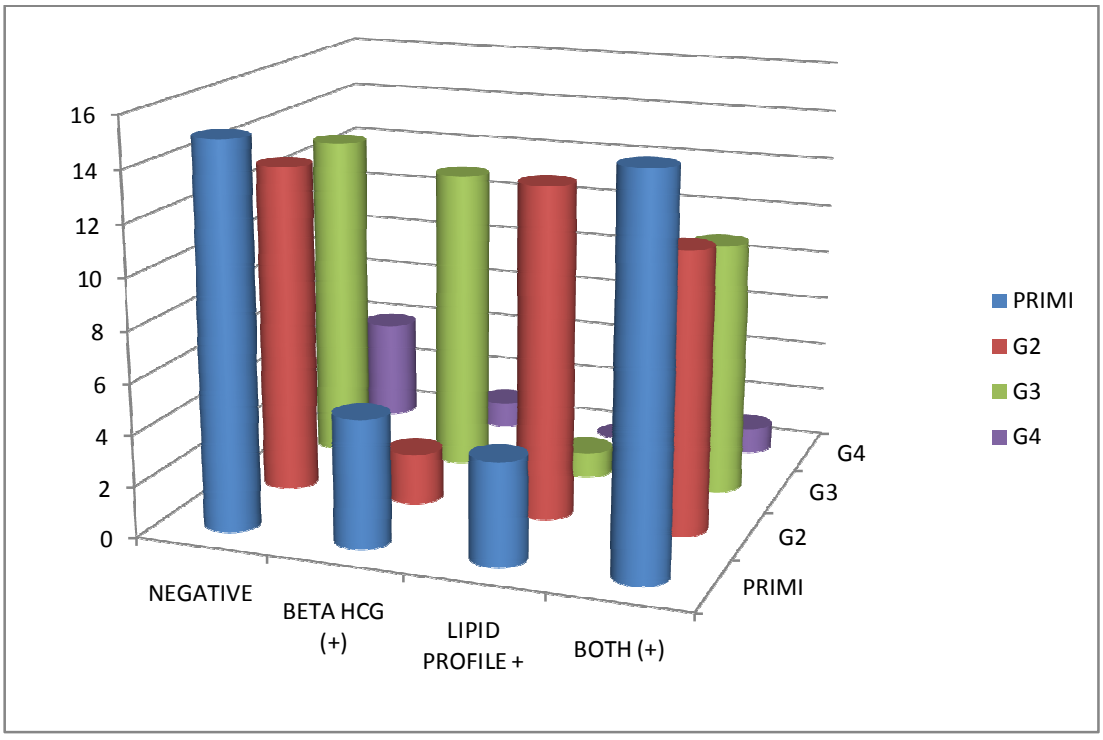


TABLE 3 : GESTATIONAL AGE

GA	14-16 wks	17-18 wks	19-20 wks
Negative	17	16	12
Beta hcg (+)	6	3	1
Lipid profile (+)	4	2	2
Both (+)	17	13	7

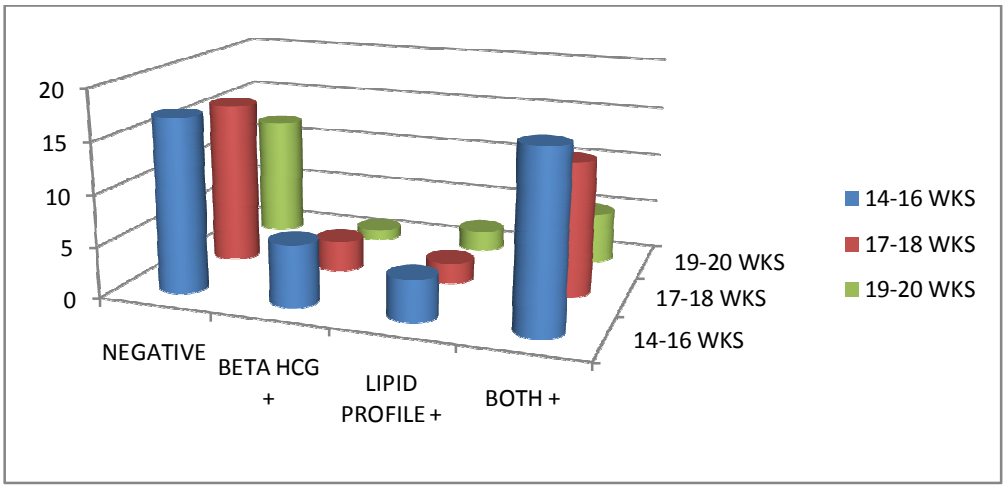
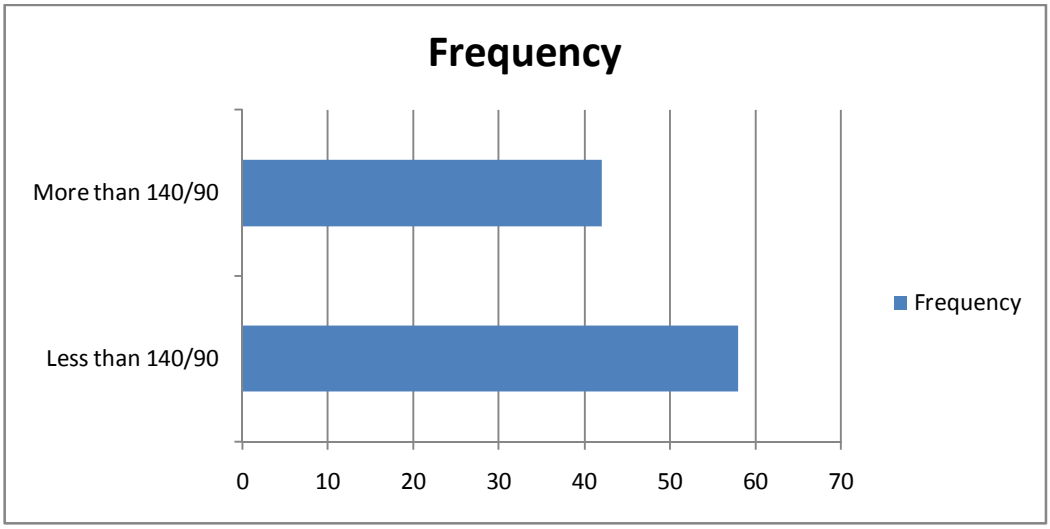


TABLE 4 : BLOOD PRESSURE

BP	Frequency
less than 140 /90	**58**
more than 140/90	**42**

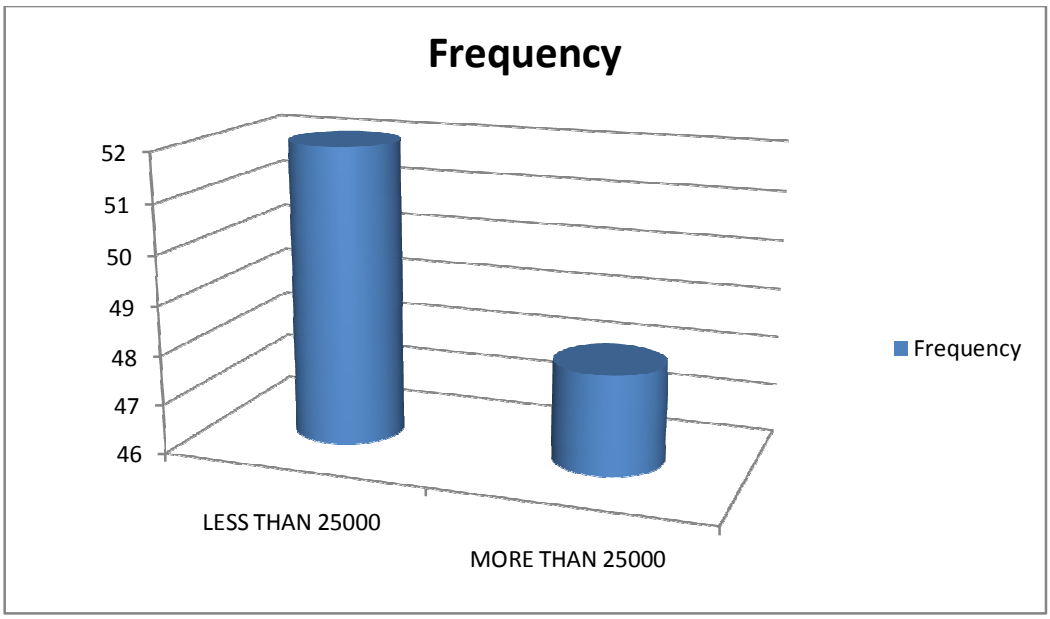


GROUP	Mean SBP with SD (mmhg)	MeanDBP with SD (mmhg)
CASES	143.76±5.84	90.28± 5.53
CONTROLS	115.8±9.02	81.2±3.84

Showing MEAN SBP ± SD of blood pressure in the study group was 143.76±5.84. MEAN DBP ±SD of blood pressure was 90.28±5.53

TABLE 5 : BETA HCG

HCG	FREQUENCY
LESS THAN 25000	52
MORE THAN 25000	48



52% had beta hcg less than 25000 & 48% had beta hcg more than 25000.

TABLE 6: CORRELATION BETWEEN HCG & BP

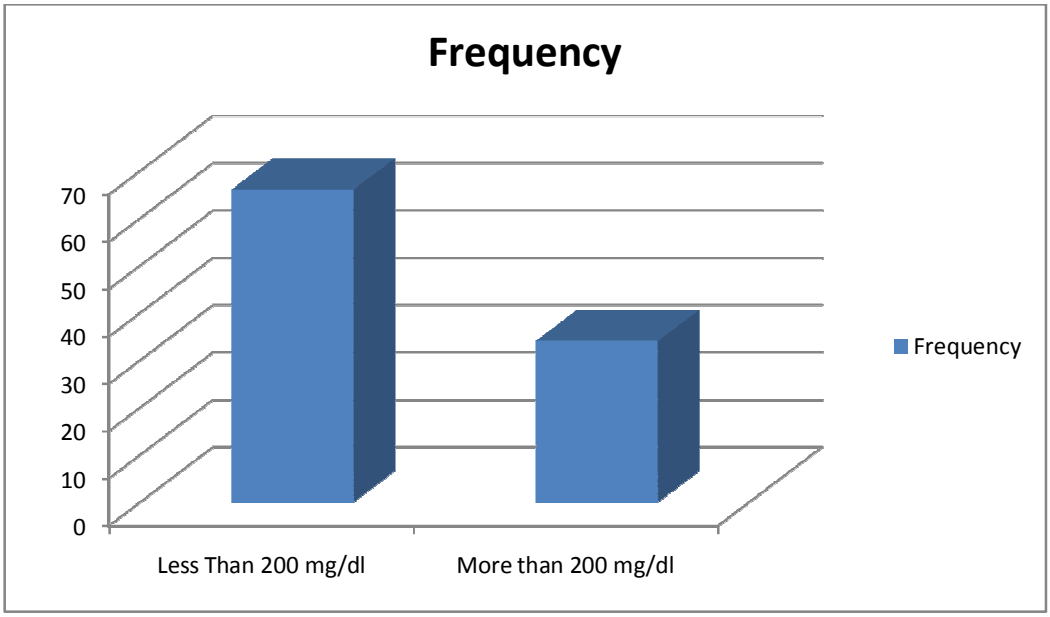
HCG	BP		SI
	less than 140 /90	more than 140/90	
Less than 25000	42	10	$x^2 = 23.056$
More than 25000	16	32	df = 1 P< 0.01 Significant

Among 100 women,52 of them had beta hcg less than 25000, among them only 10 women developed GHT. Among the 48 women who had serum beta hcg more than 25000, 16 women did not develop GHT. 32 developed GHT. Statistically significant relationship exist between elevated serum beta hcg and development of GHT.

Sensitivity	65%
Specificity	80%
Positive predictive value	86%
Negative predictive value	74%

TABLE 7: TRIGLYCERIDE VALUE

TGL	FREQUENCY
LESS THAN 200 mg/dl	66
More than 200 mg/dl	34



34% had triglycerides level more than 200 mg/dl. 66% had triglycerides level less than 200 mg/ dl.

TABLE 8: CORRELATION BETWEEN TGL & BP

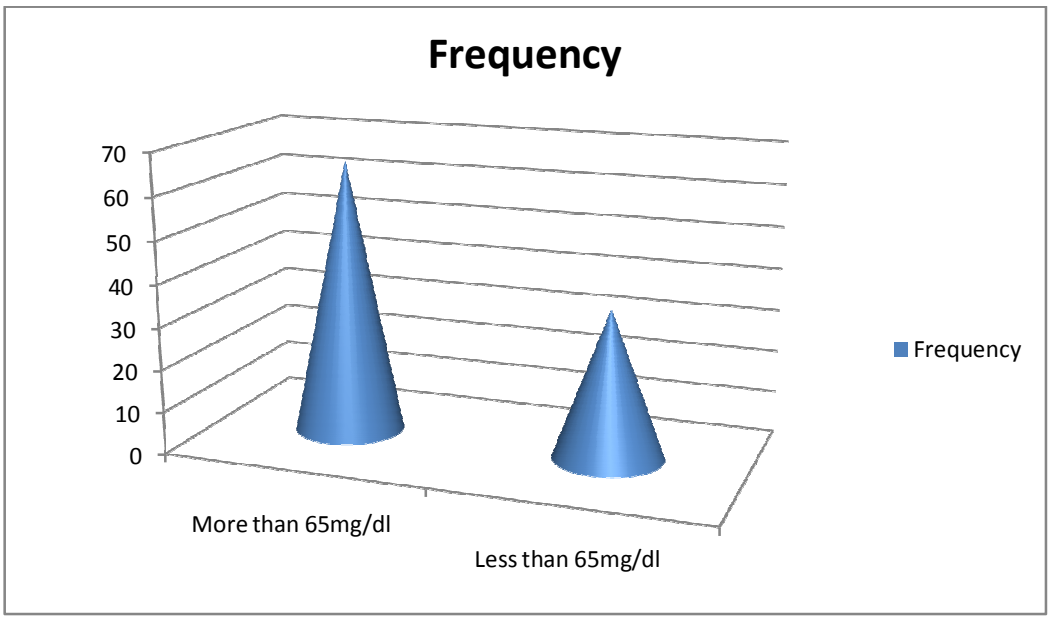
TGL	BP		Total
	less than 140 /90	more than 140/90	
Less than 200	51	15	x^2= 29.599
More than 200	7	27	df = 1 P< 0.01 Significant

Among 100 women 66 of them had triglycerides level less than 200 mg/dl, among them only 15 women developed GHT. Among the 34 women who had triglycerides more than 200 mg/dl, 7 did not develop GHT. 27 developed GHT. Statistical significant relationship exists between triglycerides and development of GHT.

Sensitivity	78%
Specificity	98%
Positive predictive value	97%
Negative predictive value	85%

TABLE 9 : HDL VALUE

HDL	Frequency
More than 65mg/dl	65
Less than 65mg/dl	35



35% had HDL less than 35% and 65% had elevated HDL

TABLE 10. CORRELATION BETWEEN HDL & BP

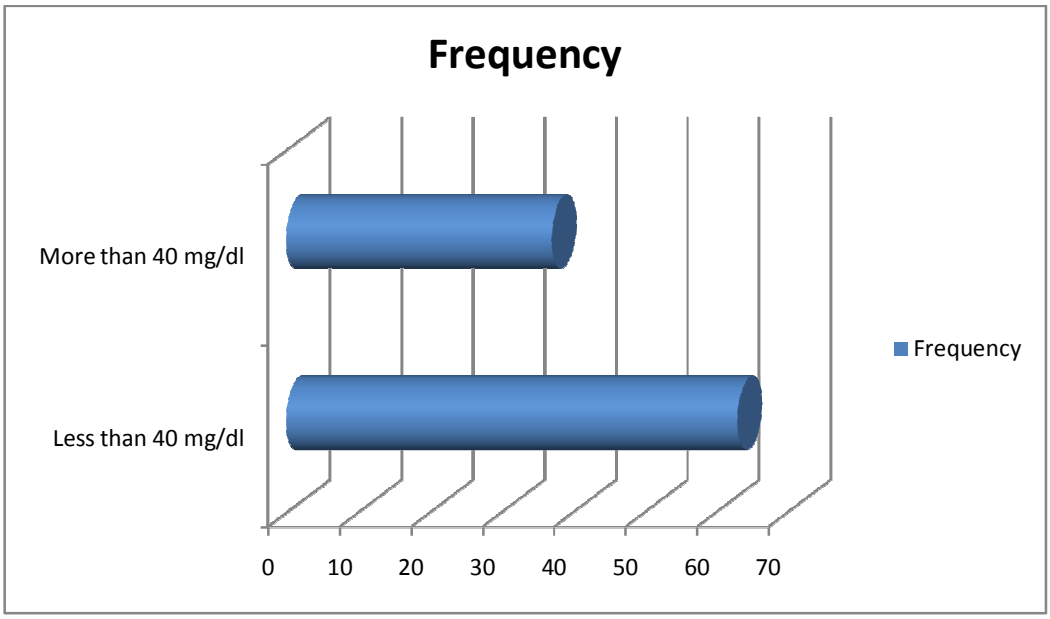
HDL	BP		Total
	less than 140 /90	more than 140/90	
more than 65	55	10	x^2= 54.005
less than 65	3	32	df = 1 P< 0.01 Significant

Among 100 women 65 of them had HDL level more than 65 mg/dl , among them only 10 of women developed GHT. Among the 35 women who had HDL less than 65 mg/dl, 3 did not develop GHT. 32 developed GHT. Statistical significant relationship exists between HDL and development of GHT.

Sensitivity	69%
Specificity	90%
Positive predictive value	86%
Negative predictive value	77%

TABLE 11 VLDL VALUE

VLDL	FREQUENCY
Less than 40 mg/dl	63
More than 40 mg/dl	37



37 % had elevated VLDL level more than 40 mg/ dl. 63% had VLDL level less than 40 mg/dl.

TABLE 12: CORRELATION BETWEEN VLDL & BP

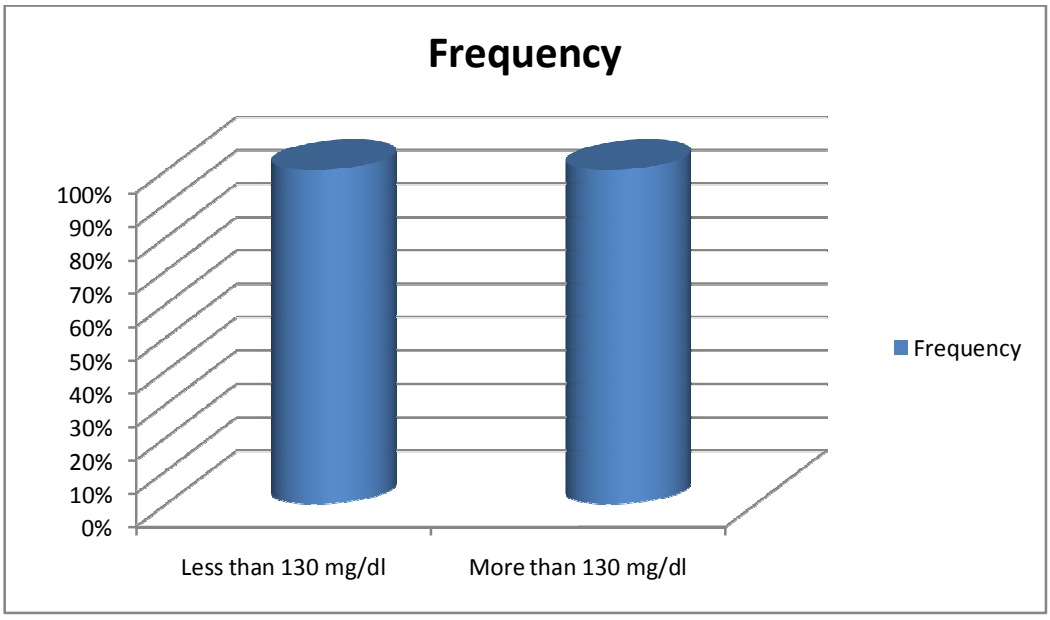
VLDL	BP		Total
	less than 140 /90	more than 140/90	
less than 40	53	10	x^2= 47.713
more than 40	5	32	df = 1 P< 0.01 Significant

Among 100 women 53 of them had VLDL level less than 40 mg/dl among them only 10 of women developed GHT. Among the 35 women who had VLDL more than 40 mg/dl, 5 did not develop GHT. 32 developed GHT. Statistically significant relationship exists between VLDL and development of GHT.

Sensitivity	69%
Specificity	92%
Positive predictive value	88%
Negative predictive value	78%

TABLE 13: LDL VALUE

LDL	frequency
Less than 130 mg/dl	61
More than 130 mg/dl	39



61 % had LDL level less than 130 mg/dl. 39% had LDL level more than 130 mg/dl

TABLE 14 : CORRELATION BETWEEN LDL & BP

LDL	BP		SI
	less than 140 /90	more than 140/90	
less than 130	55	6	x^2= 66.424
more than 130	3	36	df = 1 P< 0.01 Significant

Among 100 women 61 of them had LDL level less than 130 mg/dl among them only 6 of women developed GHT. Among the 39 women who had LDL more than 130 mg/dl, 3 did not develop GHT. 36 developed GHT. Statistically significant relationship exists between VLDL and development of GHT.

Sensitivity	78%
Specificity	98%
Positive predictive value	97%
Negative predictive value	85%

TABLE 15

CORRELATION BETWEEN β HCG & LIPID PROFILE

	Beta HCG	**TGL**	**HDL**	**VLDL**	**LDL**
Sensitivity	65%	78%	69%	69%	78%
Specificity	80%	98%	90%	92%	98%
Positive predictive value	86%	97%	86%	88%	97%
Negative predictive value	74%	85%	77%	78%	85%

TGL & LDL had high specificity (98%) and high positive predictive value (97%) than other parameters. Among them TGL & LDL can be used as a good predicting tool to identify those who developing GHT later.

TABLE 16

STRENGTH OF ASSOCIATION BETWEEN BETA HCG& LIPID PROFILE WITH GHT

PARAMETERS	RELATIVE RISK (%)
BETA HCG	3.2%
TGL	1.8%
HDL	3.2%
LDL	6%
VLDL	3.2%

For lipid profile , for one unit increase in LDL, pregnant women had 6% probability of developing GHT. For one unit increase in VLDL & Triglycerides & BETA HCG there is 3.2% and 1.8% and 3.2% increased change of developing GHT. On the contrary, with one unit increase in HDL , the women had 11.4% less chance of developing GHT

TABLE 17: FOLLOW UP STUDY

	Positive	Total no of GHT				
			6th Month	7th Month	8th Month	9th Month
β-hcg	48	32	-	-	5	27
HDL	35	32	-	-	6	26
VLDL	37	32	-	4	10	18
LDL	39	36	-	1	6	29
TGL	33	27	-	-	12	15

Among this parameter those who had elevated VLDL lipoproteins are more prone to develop early onset GHT than others.

4TH MONTH

SERUM BETA Hcg ↑ LDL ↑ VLDL ↑

9TH MONTH

BP 140 / 90

TABLE 18 :

MODE OF DELIVERY IN TOTAL POPULATION:

LN		LSCS		Assisted	Expulsion
Spontaneous	Induced	Emergency	Elective		
34	28	29	7		2

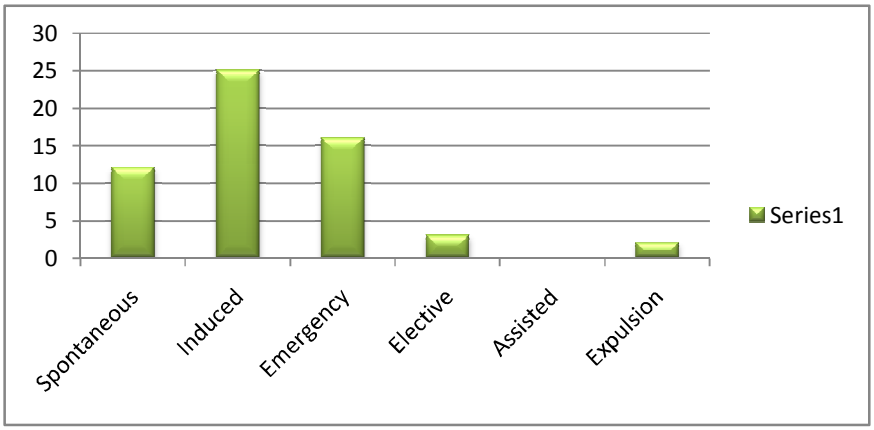
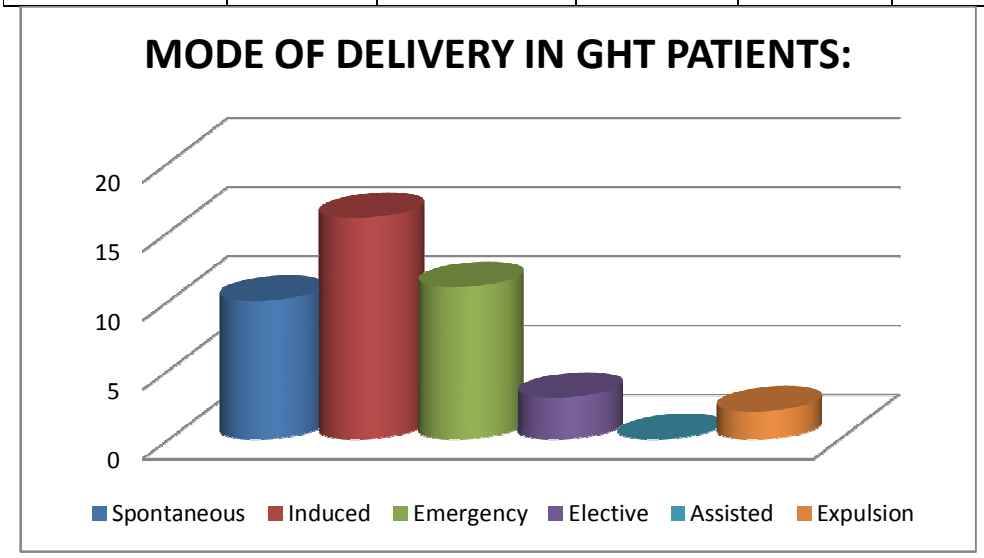


TABLE 19 MODE OF DELIVERY IN GHT PATIENTS:

LN		LSCS		Assisted	Expulsion
Spontaneous	Induced	Emergency	Elective		
10	16	11	3	0	2

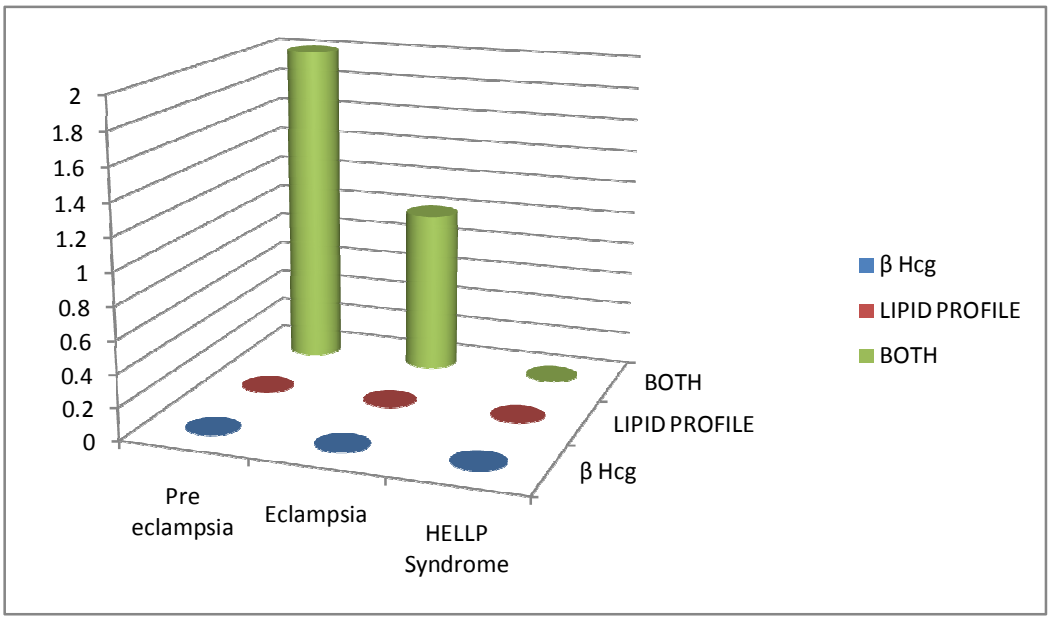


In GHT induction of labour was more common. As other causes of induction were not excluded in study, we cannot correlate significantly

TABLE 20

MATERNAL COMPLICATION

COMPLICATION	BETA HCG (+)	LIPID PROFILE (+)	BOTH
Pre eclampsia	0	0	2
Eclampsia	0	0	1
HELLP syndrome	0	0	0

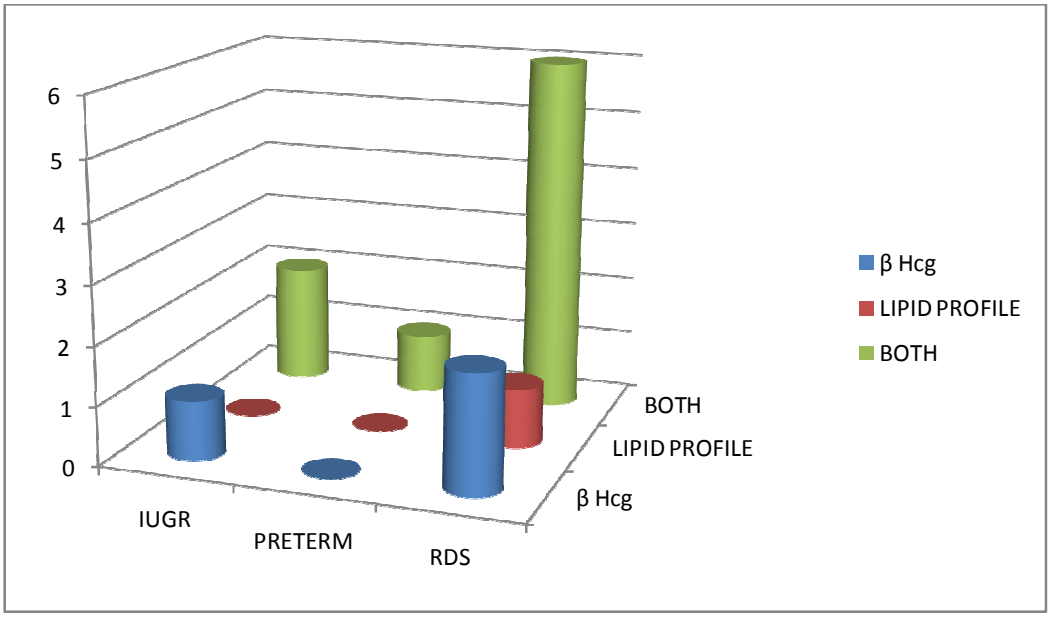


Women who had both beta HCG & lipid profile abnormalities , increased risk of developing complication than isolated abnormalities.

TABLE 21

FETAL COMPLICATION

complication	Beta HCG (+)	Lipid profile (+)	Both (+)
IUGR	1	0	2
PRETERM	0	0	1
RDS	2	1	6



Among fetal complication RDS was most common in GHT women. Women who had both beta HCG & lipid profile abnormalities had increased risk of having IUGR.

DISCUSSION

Pregnancy is the most important period in women's life, but it can be dangerous also. Hypertension and proteinuria are the important complications of pregnancy. Abnormal placentation is the one of the important pathology for the development of GHT. Because of abnormal placentation there may be increased synthesis of beta HCG.

There may be a dysregulation of lipoprotein lipase in GHT prone women, that causes elevated plasma lipid and lipoprotein levels, may induce endothelial dysfunction secondary to oxidative stress. Endothelial dysfunction is the prominent pathology, usually occurs in early trimester (8-18weeks) but signs and symptoms occur in late trimester.

In this study serum beta HCG estimated in early second trimester, women with elevated levels, categorized under high risk group. So it is easy to identify the high risk women and kept under regular follow up. It is help in preventing development of complication in GHT.

Study conducted in OP population in Raja Mirasudar Hoapital, Thanjavur Medical College, Thanjavur. It is a prospective study, conducted on 100 pregnant women who attended the antenatal OPD. All the patients screened for BETA HCG & lipid profile in early second trimester between (14-20 weeks) and followed up till their delivery.

Normally GHT is more common in teenage pregnancies. In our study most of the GHT patient within the age group between 26-30 years with standard deviation of 24.48 ± 2.08 years.

Normally GHT more common in primi gravid, in our study also more number of GHT cases were primi gravid.

Blood pressure (140/90 mm of Hg) was used as a cut off for GHT. 58% had less than that above value, 42% had blood pressure more than 140/90 mm of Hg. GHT cases had mean SBP with SD of about 143.76±5.84 and mean DBP with SD was 90.28± 5.53.

Regarding beta HCG 25000 mIU/l used as a cut off value. In 100 population 52% had less than 25000 beta HCG & 48% had more than 25000 beta HCG value.

In our study among 100 women, 48 of them had elevated beta HCG value, among them 32 of them developed GHT. 52 of them had beta HCG less than 25000, among them only 10 women developed GHT. With that value statistical significant relationship exist between elevated beta HCG and development of GHT with p value <0.01.

This is in accordance with the study done by the Yaron et al[21] & Ellis p et al & Hijam danina et al , they also showed that there is significant increase in serum beta HCG in early second trimester and development of GHT .

In our study beta HCG showed 80% specificity & 86% positive predicting value. Similar results were obtained in Yaron et al. They shown that beta HCG had high positive predictive value of about 88% .They concluded with that predictive value, serum beta HCG can be used as a predictor for GHT.

In our study strength of association between beta HCG and GHT is about 3.2%. In Vidyabati et al [25](2003) they proven that, for one unit increase in the beta HCG, pregnant women had 3.7% increasing chance of GHT.

In our study among 100 women, 34 of them had elevated triglycerides value, among them 22 of them developed GHT. 66 of them had triglycerides less than 200, among them only 15 women developed GHT. With that value statistical significant relationship exist between elevated triglycerides and development of GHT with p value <0.01.

In Clause T[18], (1998) & Djurovic et al[21] (2001) found that dyslipidemia seems to be more efficient marker in predicting GHT at early second trimester with statistical significant (p <0.05). In Clause T did a study in 1998 in100 pregnant women, in that 54 % had elevated triglycerides, among them 48% developed GHT with statistical significant value.

In our study elevated triglycerides value had 98% high specificity 0& 97% positive predictive value. In Pouta et al (2000) found that

significant correlation between the elevated triglycerides and development of GHT with 96% specificity. Clausen et al[26] concluded that hypertriglycedemic dyslipidemia before 20 weeks of gestation had increased relationship with early onset GHT . They had a significant p value of about <0.01 and high positive predictive value (92%)

In our study strength of association between triglycerides and GHT is about 1.8%. In Vidyabati et al[25] (2003) they proven that, for every one unit increase in the triglycerides, pregnant women had 2.0% increasing chance of GHT.

In our study among 100 women, 35 of them had reduced HDL value, among them 32 of them developed GHT. 65 of them had HDL more than 65, among them only 10 women developed GHT. With that value statistical significant relationship exist between reduced HDL and development of GHT with p value <0.01.

Turpin et al [42](2001) did a study in 150 women & found that women who had elevated HDL value, have a reduce risk of GHT. Islam et al (1998) revealed that women with reduced HDL value more prone to develop GHT with a statistical significant value (p<0.05).

In our study HDL had 90% specificity & 86% positive predictive value. Similar results were obtained by Lima et al (1999) with 92% specificity & 88% positive predictive value.

In our study strength of association between increased HDL and preventing GHT is about 3.2%. In Vidyabati et al[25] (2003) they prove that, for one unit increase in the HDL, a pregnant women had 3.7% less chance of GHT.

In our study among 100 women, 37 of them had elevated VLDL value, in which 32 of them developed GHT. 63 of them had VLDL less than 40, among them only 10 women developed GHT. With that value statistical significant relationship exist between elevated triglycerides and development of GHT with p value <0.01.

In 1998 Taital mikic researchers & De et al [44](2004) they found that there is a significant rise in VLDL value in GHT women. They did Meta analysis study in 2004; they concluded that elevated VLDL is a common finding in GHT women, in all study. They had a statistical significant value between elevated VLDL and GHT women (p<0.001).

In our study VLDL had 92% specificity, this is supported by FranZ H, Wender D (2002), they proven that elevated VLDL had 90% specificity & positive predictive value, with that they agreed that VLDL is the one of good predictor for GHT.

In our study among 100 women, 39 of them had elevated LDL value, among them 36 of them developed GHT. 61 of them had LDL less than 130, among them only 6 women developed GHT. With that value

statistical significant relationship exist between elevated triglycerides and development of GHT with p value <0.01.

In 2005 Cekman et al[28] did a study in 150 pregnant women. In that 96 of them had elevated LDL more than 130mg/dl, among them 78 developed GHT. 54 of them had normal LDL value. In this only 10 developed GHT. With this result they proved very highly significant relationship between LDL and GHT.

In our study strength of association between LDL& VLDL with GHT was 6% & 3.2% respectively. Wakatsuki et al [40](2001) had proven that similar association between the LDL& VLDL with GHT . For every one unit increase in LDL & VLDL 6.8% & 4.5% there is increase risk of developing GHT in pregnant women respectively.

Induction of labour is more common in GHT. This also happened in our study, but there is no statistical significance was obtained in our study. This may be due to small sample size, variation in the gravida in total study population.

In our study 2 of them had severe pre eclampsia & 1 of them had eclampsia. Women with severe preeclampsia had high levels both beta HCG & lipid profile particularly TGs. This was supported by Turpin et al[42] in the year 2003 made a study with lipid changes in pregnant women associated with the development of gestational hypertension. They concluded that women with elevated triglycerides level (> 3.0 MoM) had

increased chances (17%) of developing GHT and early onset severe pre eclampsia.

- In our study fetal complications like IUGR and RDS are more common in GHT patients. This supported by Gonen et al & Valiient et al , they found that there is increase risk of development of IUGR & RDS more common in GHT than control groups. In the year 2003Vallient et al [40]demonstrated that free beta HCG was a marker of high risk for GHT & associated with small for gestational age .They did a study in 192 pregnant women. Women with beta HCG > 3.5 MOM is taken as study group. In 192, 87 of them had elevated beta HCG. In the study group 24 of them developed GHT in third trimester. In which 12 of them had IUGR baby .They also found that women with elevated serum beta HCG were associated with increased risk of IUGR baby with statistical significance value ($p < 0.01$).

CONCLUSION

Even in this era of advanced medical knowledge what we don't know far outweighs what we know. The case is no different with GHT, a leading killer of mothers worldwide. Despite one of the top causes for maternal mortality and morbidity very little is known about its aetiology. Many studies were conducted to determine to exact sequence of events behind its presentation. Recent studies have found out that the clinical manifestation of GHT is proceeded way back by biochemical and pathological changes in the body. This is the reason why all modes of treatment except delivery are only palliative.

If we can detect these changes before hand a major share of maternal mortality can be prevented. The level of beta HCG and lipid profile are two factors strongly associated with development of GHT. These can be used as "POWERFUL PREDICTIVE TOOL" for obstetrician for early identification and expert management. Then close monitoring of maternal and fetal status of identified cases can be done in tertiary care centre like our institution resulting in a good maternal and perinatal care

SUMMARY

A Study of serum beta HCG and lipid profile in a early second trimester is a predictor of GHT.

This study was done at Thanjavur medical college , Thanjavur.

- ✓ In total 100 women, initial beta HCG and lipid profile evaluation was done in early second trimester.
- ✓ Those who have elevated value taken as cases and others included in control population.
- ✓ Elevated values in cases were compared with GHT population, showed statistically significant ($p<0.01$)

The present study showed elevated beta HCG and lipid profile in GHT, suggesting the role of abnormal placentation and endothelial dysfunction due to lipoprotein lipase in the aetiology of GHT , though the exact mechanism remains to be elucidated.

The strength of association between these factors and GHT are very much significant. So it can be used as a predictor of GHT. These factors are very useful in the earlier period of gestation in the screening of GHT.

In this study there is no maternal mortality, CVA, severe acute renal failure, HELLP syndrome. With the regular monitoring I had 2 cases of severe pre eclampsia and one eclampsia. Regarding the perinatal outcome incidence of IUGR and RDS are more common in GHT.

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ANNEXURE-1

CONSENT FORM

I ……..exercising my free power of choice here by give consent to participate in the study conducted by **Dr.V.MUNIRA** Post graduate in Department of Obstetrics and Gynaecology, Govt. Raja Mirasudhar Hospital, Thanjavur – 613001. I have been given full explanation in my own language about the purpose of study and the investigations for any complications with co-morbid conditions.

Signature of the participant

Place:

Date:

PROFORMA

NAME:

AGE:

OP NO:

DIAGNOSIS:

PAST H/O: DM/ HT/TB/EPILEPTIC/HEART DISEASE/THYROID PROBLEM

FAMILY H/O

OBSTETRIC H/O:

Mode of delivery: FTNVD/ LSCS

Birth weight:

Postnatal events:

GENERAL EXAMINATION:

HT: WT : BMI: BP:

Anemic/ PE/ icteric/ lymphadenopathy

P/A:

U/A:

SERUM BETA HCG:

LIPID PROFILE:

1. TGL
2. HDL
3. LDL
4. VLDL

FOLLOW UP:

	6th month	7th month	8th month	9th month
PE				
U/A				
BP				

MODE OF DELIVERY:

FTNVD: spontaneous/ induced
FTLSCS: elective/emergency
ASSISTED:

SPONTANEOUS EXPULSION:

MATERNAL COMPLICATION:

1. Severe pre eclampsia:
2. HELLP syndrome:
3. Eclampsia:

FETAL COMPLICATION:

1. IUGR:
2. RDS:
3. LBW

S. No	Name	Age	DP No.	obstetric	GA	Past H/O	Previous Obstric H/O			General			BP	Lab		P/A	Sem B.hu	TC	TOTAL CHOLESTEROL		
							MOD	BW	PN events	A	PE	I		HB	UA				HOL	LOL	VLDL
1	JASMINE DELPHI	30		G3P1L1A1	16	_	LSCS	3	_	N	N	N	110/70	10	_	16	26000	>	>	N	N
2	ABINAYA	20		PRIMI	14	GDM	_	_	_	N	N	N	120/60	9.6	_	14	28000	>	<	N	N
3	MARIYAMMAL	26		G2P1L1	14	_	_	_	_	N	N	N	110/70	9.5	_	14	21000	N	N	N	N
4	RADHALAKSHMI	33		G3P2L2	15	_	2FTNVD	2.5/2.5	_	N	yes	N	120/70	10	_	14	31000	>	<	>	N
5	KARTHIKA	30		G2A1	15	_	0	_	_	N	N	N	110/70	9.8	_	14	23000	N	N	N	N
6	AROKIYASHEELA	26		G2P1L1	17	_	FTNVD	_	_	N	N	N	120/60	9.6	_	16	25600	>	>	N	N
7	RUTHNISHA	25		PRIMI	18	_	0	_	_	N	N	N	110/70	9.5	_	18	31000	N	N	N	N
8	LAKSHMI	28		G3P2L2	19	_	1FTND/1LSCS	2.8/2.6	_	N	N	N	120/60	9.7	_	18	26000	<	>	N	>
9	MAHALAKSHMI	27		G3P1L1A1	19	_	FTNVD	_	_	MA	N	N	110/80	9.5	_	18	22000	N	N	N	N
10	RANI	27		G3P1L1A1	14	PIH	0	_	_	N	N	N	100/70	9.5	_	14	21000	>	<	>	N
11	DEVI	19		PRIMI	14	_	0	_	_	N	N	N	110/80	9.3	_	14	26000	N	N	N	N
12	SUSEELA	21		G2A1	20	_	0	_	_	N	N	N	110/60	9.4	_	20	23000	N	N	N	N
13	SUTHANNATHIYA	29		G3P2L2	20	_	2FTNVD	2.6/2.5	_	N	N	N	120/80	9.5	_	20	28000	>	N	N	<
14	DILSATHBEGUM	28		PRIMI	15	_	0	_	_	N	N	N	110/70	9.5	_	14	23000	N	N	N	N
15	ANJUPOON	26		G2P1L1	17	PIH	LSCS	_	_	N	N	N	110/60	9.6	_	16	26000	>	<	N	>
16	ASHIFAHANIFER	26		G2A1	18	_	0	_	_	N	N	N	100/60	9.4	_	18	23000	N	N	N	N
17	LAKSHMIPRIYA	29		G3P1L1A1	16	_	0	2.5	_	N	N	N	110/70	9.6	_	18	24660	N	N	N	N
18	SHANMUGALAKSHMI	23		PRIMI	16	_	_	_	_	N	N	N	120/60	9.3	_	16	23000	>	<	N	N
19	THAHIRADILFAR	23		PRIMI	16	_	_	_	_	N	N	N	110/70	9.5	_	16	30000	>	<	N	N
20	PANIMALAR	25		G2P1L1	14	_	LSCS	3	_	N	N	N	100/60	9.5	_	14	21500	N	N	N	N
21	VASUKI	28		G2A1	14	_	_	_	_	N	N	N	120/80	10	_	14	21000	N	N	N	N
22	DHARANIJAYASHREE	26		PRIMI	14	_	_	_	_	N	N	N	110/70	9.8	_	14	21500	N	N	N	N
23	MALATHI	30		G4P1L1A2	15	PIH	FTNVD	2.614	_	N	N	N	120/80	9.6	_	14	31000	>	<	N	N

S. No	Name	Age	DP No.	obstetric	GA	Past H/O	Previous Obstric H/O			General			BP	Lab		P/A	Sem B.hu	TC	TOTAL CHOLESTEROL		
							MOD	BW	PN events	A	PE	I		HB	UA				HOL	LOL	VLDL
24	NALINI	21		PRIMI	16	_	_	_	_	MA	N	N	110/70	9.6	_	16	22000	N	N	N	N
25	SUNANDHA	24		G2A1	18	_	_	_	_	N	N	N	120/80	9.6	_	18	26000	N	N	N	N
26	SELVAPRIYA	21		PRIMI	16	_	_	_	_	N	N	N	110/70	9.5	_	16	24000	N	N	N	N
27	DEEPALAKSHMI	26		G2P1L1	18	_	FTNVD	2.5	_	N	YES	N	120/60	9.6	_	18	28000	N	N	N	N
28	KANJANA	23		PRIMI	20	_	_	_	_	N	N	N	100/60	9.5	_	18	23000	>	N	>	N
29	KAVITHA	25		G3P1L1A1	20	_	FTNVD	3	_	N	N	N	110/70	9.4	_	20	24000	N	N	N	N
30	VANITHA	19		PRIMI	18	_	_	_	_	N	N	N	120/80	9.6	_	18	28000	N	N	N	N
31	YOGALAKSHMI	19		PRIMI	16	_	_	_	_	N	N	N	110/70	9.7	_	16	23000	>	>	N	N
32	AMUDHA	24		G2P1L1	17	_	FTLSCS	2.8	_	N	N	N	120/80	9.5	_	16	23000	N	N	N	N
33	MALLIGA	20		PRIMI	15	_	_	_	_	N	N	N	110/70	9.4	_	14	22000	N	>	N	N
34	MARAGATHAM	21		PRIMI	14	_	_	_	_	N	N	N	120/70	9.6	_	14	21000	N	N	N	N
35	SATHYAVANI	21		G2A1	18	_	_	_	_	N	N	N	110/80	9.7	_	14	22000	N	N	N	>
36	AMIRTHAVALLI	26		G3P1L1A1	19	_	FTNVD	2.9	_	N	N	N	100/60	9.5	_	18	26000	N	N	N	N
37	SARASWATHY	30		G3P2L2	16		2FTND	2.5,2.5		N	N	N	110/70	9.4	_	16	23000	>	<	N	N
38	SHANMUGAM	26		G2A1	14	_	_	_	_	N	N	N	110/70	9.6	_	14	22000	N	N	N	N
39	JEYAPRADHA	26		PRIMI	15	_	_	_		N	N	N	110/60	9.6	_	14	22000	N	N	N	N
40	MARIYAMMAL	30		G4P1LIA2	17	_	FTLSCS	3		N	N	N	110/70	9.4	_	16	24000	<	>	N	N
41	ABIRAMI	33		G3P2L2	17	_	1LSCS 1FTNVD	3, 2.2		N	N	N	100/60	10	_	16	25500	N	N	N	N
42	KOLANJIYAM	30		G3P1L1A1	19	_	1FTNVD	3		MA	N	N	120/70	10	_	18	25500	N	N	N	N
43	KOKILA	19		PRIMI	20	_	_	_		N	N	N	110/70	10	_	20	26000	>	<	>	N
44	GOMATHY	21		G2P1L1	19	_	1LSCS	2.4		N	N	N	120/80	10.7	_	18	24000	N	N	N	N
45	SHANTHI	28		G3P2L2	18	_	2 LSCS	2.6,2.9		N	N	N	110/70	10.5	_	18	23300	N	N	N	N
46	KUSHPOO	22		PRIMI	14	_	_	_		N	N	N	120/70	10.8	_	14	27000	>	>	>	N

S. No	Name	Age	DP No.	obstetric	GA	Past H/O	Previous Obstric H/O			General			BP	Lab		P/A	Sem B.hu	TC	TOTAL CHOLESTEROL		
							MOD	BW	PN events	A	PE	I		HB	UA				HOL	LOL	VLDL
47	JHANANI	26		G2A1	20	_	_	_		N	N	N	110/70	9	_	20	25000	N	N	N	N
48	JEYASHREE	28		G2P1L1	14	_	FTNVD	2.7		N	N	N	100/60	9.6	_	14	23400	N	N	N	N
49	DHIVYAPRIYA	30		G3P1L1A1	15	_	FTNVD	2.9		N	N	N	120/70	9.7	_	14	24300	>	<	N	N
50	BABYNISHA	20		PRIMI	15	_	_	_		N	_	N	120/60	9.8	_	14	28000	N	N	N	N
51	DURGALAKSHMI	28		G3P1L1A1	18	_	FTNVD	2.6		N	N	N	110/70	9.6	_	18	21000	N	N	N	N
52	JEMIR FATHIMA	26		G2A1	19	APPENDIX	_	_		N	N	N	100/70	9.6	_	18	22500	N	N	N	N
53	BRISILA ROSELIN MAR	29		G3P2L2	17	_	2 LSCS	_		N	N	N	120/80	9.5	_	16	23000	N	N	N	N
54	ALAGUPRIYA	22		G2A1	17	_	_	_		N	N	N	110/70	9.4	_	16	26000	>	>	N	N
55	AMBIKA	20		PRIMI	16	_				N	N	N	120/70	9.2	_	16	26000	>	<	>	N
56	KAVITHA	26		G3P1L1A1	14	_	FTNVD	3		N	N	N	110/70	9.6	_	14	21500	N	N	N	N
57	GOMATHY	25		G2A1	16	GDM				N	N	N	120/80	9.4	_	18	22000	N	N	N	N
58	SHANTHI	31		G3P2L2	15	_	2 FTNVD	3,3.2		MA	N	N	110/60	9.6	_	14	23000	N	N	N	N
59	MANGALAM	22		PRIMI	14	_				N	N	N	100/60	9.4	_	14	29000	>	<	>	>
60	SHARMILA DEVI	28		G3P1L1A1	20	_	LSCS	3.6		N	N	N	120/80	9.6	_	20	24000	N	N	N	N
61	MALARVIZHI	34		G4P1L1A2	20	_	LSCS	1.8		N	N	N	120/80	9.4	_	20	30000	>	<	>	N
62	DURGADEVI	30		G3P2L2	15	_	2 FTNVD	2,2.2		N	N	N	110/70	9.6	_	14	25000	N	N	N	N
63	ANBUSELVI	26		PRIMI	16	_				N	N	N	120/60	9.8	_	16	24600	N	N	N	N
64	KALAIARASI	27		G2P1L1	18	_	FTNVD	3		N	N	N	110/70	9.6	_	18	26000	>	<	N	N
65	MAGAMAYEE	26		G2A1	17	_				N	N	N	120/60	9.5	_	16	24000	N	N	N	N
66	RADHIKA	25		G4P1L1A2	17	_	FTNVD	3		N	N	N	110/70	9.4	_	16	23000	N	N	N	N
67	SULTAN NACHAIR	20		PRIMI	19	_				N	N	N	120/60	9.3	_	16	20000	>	<	N	N
68	MARY JENNIFER	29		G3P2L1	20	IUD	FTNVD	1.9,2.5		N	N	N	110/80	9.6	_	20	22000	N	N	N	N
69	ASHA BABY	28		G3P1L1A1	18	_	LSCS	2.5		N	N	N	120/80	9.4	_	18	21000	N	N	N	N

S. No	Name	Age	DP No.	obstetric	GA	Past H/O	Previous Obstric H/O			General			BP	Lab		P/A	Sem B.hu	TC	TOTAL CHOLESTEROL		
							MOD	BW	PN events	A	PE	I		HB	UA				HOL	LOL	VLDL
70	PANCHAVARNAM	30		G3P1L1A1	16	_	FTNVD	2.4		N	N	N	120/70	9.3	_	16	26000	>	>	N	>
71	KANAGA	19		PRIMI	18	_				N	N	N	120/80	9.7	_	18	20000	N	N	N	N
72	PRIYADARSHINI	24		G2P1L1	20	_	FTNVD	3.1		N	N	N	120/70	9.3	_	18	25000	>	<	N	N
73	MANGAYARKARASI	26		G2P1L1	20	_	LSCS	3.1		N	N	N	100/60	9.2	_	18	23200	N	N	>	N
74	ANITHA	20		PRIMI	20	_				N	N	N	120/70	9.6	_	18	26000	N	N	N	N
75	SUNDARI	26		G3P1L1A1	18	_	FTNVD	2.6		N	N	N	110/60	9.7	_	18	24000	>	<	N	N
76	NAZRIN BANU	30		G3P2L2	16	_	2 FTNVD	2.1,2.5		N	N	N	120/70	9.8	_	16	24300	N	N	N	N
77	MATILDA	20		PRIMI	17	_				N	N	N	110/60	9.4	_	16	25000	N	N	N	N
78	AASAIVALLI	26		G3P1L1A1	19	CVT	FTNVD	3		N	N	N	120/70	9.6	_	18	23200	>	>	N	N
79	AFREINSITHIHA	27		G2P1L1	19	_	LSCS	3.2		MA	N	N	120/60	9.6	_	18	25000	N	N	N	N
80	AARTHI	20		G2A1	15	_				N	N	N	100/60	9.4	_	14	30000	>	<	N	N
81	VELANKANNI	26		G2P1L1	18	_	FTNVD	3		N	N	N	110/70	9.6	_	16	26000	N	N	N	N
82	ABIRAMI	27		G3P1L1A1	15	_	FTNVD	2.5		N	N	N	120/80	9.5	_	14	24000	>	>	>	>
83	KAMALA	30		G4P2L2A1	16	_	FTNVD	2.1,2.3		N	N	N	110/70	9.4	_	16	30000	N	N	N	N
84	BARAKATH NISHA	32		G3P2L2	15	GHT	FTNVD	2.5,2.5		N	N	N	120/80	9.7	_	14	26000	N	N	N	N
85	NIRMALA	30		G2A1	17	_				N	N	N	120/80	9.4	_	16	24000	>	<	N	N
86	RANJITHA	21		PRIMI	18	_				N	N	N	120/80	9.6	_	18	22300	N	N	N	N
87	PUNITHAVALLI	26		G2P1L1	18	_	LSCS	3		N	N	N	130/80	9.4	_	18	26000	N	N	N	N
88	THINGALAVAL	24		PRIMI	15	_				N	N	N	110/70	9.6	_	14	25000	N	N	N	N
89	DHARSHINI	21		PRIMI	15	_				N	N	N	120/80	9.8	_	14	23000	N	N	N	N
90	DHANYA	20		PRIMI	16	_				N	N	N	120/80	9.4	_	16	22000	N	N	N	N
91	KURINJIPOO	26		G2P1L1	18	GDM	LSCS	3.5		N	N	N	120/80	9.8	_	18	25000	>	<	N	N
92	TAMILARASI	30		G3P1L1A1	16	_	LSCS	3		N	N	N	110/80	9.4	_	16	26000	N	N	N	N

S. No	Name	Age	DP No.	obstetric	GA	Past H/O	Previous Obstric H/O			General			BP	Lab		P/A	Sem B.hu	TC	TOTAL CHOLESTEROL		
							MOD	BW	PN events	A	PE	I		HB	UA				HOL	LOL	VLDL
93	RADHIKA	26		PRIMI	16	_				N	N	N	120/80	9.5	_	16	25000	N	N	N	N
94	MUTHULAKSHMI	25		G2A1	15	_				N	N	N	110/70	9.2	_	14	24000	N	N	N	N
95	MAHALAKSHMI	25		PRIMI	15	_				N	N	N	120/80	9.6	_	14	25000	N	N	N	N
96	RAJINISHREE	23		PRIMI	14	_				N	N	N	110/70	9.4	_	14	25000	>	<	>	N
97	JASMNE PRABHA	23		G2P1L1	15	PIH	FTNVD	2.5		N	N	N	120/80	9.8	_	14	28000	N	N	N	N
98	ACQUINES RANI	30		G3P2L2	15	GDM	FTNVD	2.6,2.8		N	N	N	110/70	9.2	_	14	26000	>	<	N	N
99	ILAKKIYA	28		G3P1L1A1	18	_	LSCS	3.5		N	N	N	120/80	9.4	_	16	27000	N	N	N	N
100	RAMYA	21		PRIMI	18	_				N	N	N	110/70	9.2	_	16	24500	N	N	N	N

S. No	Name	6 th Month			7 th Month			8 th Month			9 th Month			Mode of delivery				Spantanous			Material			Fetal complicaiton			
		PE	BP	UA	PE	BP	U/A	PE	BP	U/A	PE	BP	U/A	SPO	I	El.lscs	EM	A	D	assisted	PE	EC	Help	IUGR	PRETERM	RDS	
1	JASMINE DELPHI	PE	110/70	_	PE	110/70	NIL	PE	110/70	NIL	PE	130/90	NIL				YES										
2	ABINAYA	_	120/80	_	PE	120/80	NIL	PE	100/70	NIL	PE	140/90	NIL		YES											YES	
3	MARIYAMMAL	_	110/70	_	_	120/60	NIL	_	110/70	NIL	_	120/80	_	YES													
4	RADHALAKSHMI	_	120/80	_	_	120/70	NIL	PE	120/80	NIL	PE	130/90	NIL		YES											YES	
5	KARTHIKA	_	110/70	_	_	110/70	NIL	_	140/80	NIL	_	110/80	_	YES													
6	AROKIYASHEELA	_	110/70	_	_	110/70	NIL	_	130/80	NIL	_	120/70	_				YES										
7	RUTHNISHA	_	110/70	_	_	120/80	NIL	_	120/80	NIL	_	110/80	_	YES													
8	LAKSHMI	_	120/80	_	_	110/70	NIL	PE	110/70	NIL	PE	130/90	NIL		YES		YES										
9	MAHALAKSHMI	_	110/70	_	_	120/70	NIL	_	120/80	NIL	_	120/80	_				YES										
10	RANI	_	120/80	_	_	110/70	NIL	PE	110/70	NIL	PE	140/90	NIL	YES													
11	DEVI	_	110/70	_	_	120/80	NIL	_	120/80	NIL	_	110/70	_	YES													
12	SUSEELA	_	120/80	_	_	110/70	NIL	_	110/70	NIL	_	120/80	_				YES								YES/LATE		
13	SUTHANNATHIYA	_	110/70	_	_	110/80	NIL	_	120/80	NIL	_	140/90	NIL		YES												
14	DILSATHBEGUM	_	120/80	_	_	110/70	NIL	_	120/80	NIL	_	110/70	_	YES													
15	ANJUPOON	_	110/70	_	_	120/70	NIL	PE	140/90	NIL	PE	140/90	NIL				YES									YES	
16	ASHIFAHANIFER	_	110/80	_	_	110/80	NIL	_	120/80	NIL	_	110/70	YES	YES													
17	LAKSHMIPRIYA	_	110/70	_	_	110/70	NIL	_	110/70	NIL	_	110/80	_				YES										
18	SHANMUGALAKSHMI	_	120/80	_	_	120/80	NIL	PE	120/80	NIL	PE	140/90	NIL		YES												
19	THAHIRADILFAR	_	100/60	_	_	110/70	_	_	130/90	NIL	PE	140/90	YES		YES												YES
20	PANIMALAR	_	110/70	_	_	120/70	_	_	120/70	NIL	_	120/70	_				YES										
21	VASUKI	_	110/80	_	_	120/80	_	_	110/60	NIL	PE	120/70	_				YES								YES/MILD		
22	DHARANIJAYASHREE	_	120/80	_	_	110/70	_	_	130/80	NIL	PE	130/90	YES		YES												
23	MALATHI	_	120/80	_	_	130/80	NIL	_	130/80	NIL	_	130/90	YES		YES												YES

S. No	Name	6 th Month			7 th Month			8 th Month			9 th Month			Mode of delivery				Spantanous			Material			Fetal complicaiton			
		PE	BP	UA	PE	BP	U/A	PE	BP	U/A	PE	BP	U/A	SPO	I	El.lscs	EM	A	D	assisted	PE	EC	Help	IUGR	PRETERM	RDS	
24	NALINI	_	110/70	_	_	120/70	_	_	110/70	NIL	_	110/70	_				YES										
25	SUNANDHA	_	100/60	_	_	110/80	_	_	120/70	NIL	_	120/70	_	YES													
26	SELVAPRIYA	_	110/70	_	_	110/80	_	PE	130/80	YES	PE	130/90	YES		YES												
27	DEEPALAKSHMI	_	110/80	_	_	110/70	_	_	110/70	NIL	_	110/70	_	YES													
28	KANJANA	_	110/70	_	_	120/80	_	_	110/70	NIL	_	130/90	NIL				YES										
29	KAVITHA	_	110/80	_	_	120/80	_	_	110/70	NIL	_	110/70	_	YES													
30	VANITHA	_	110/70	_	_	110/70	_	_	120/80	NIL	_	120/80	_	YES													
31	YOGALAKSHMI	PE	120/80	_	PE	120/80	_	PE	110/70	NIL	PE	130/90	NIL				YES										
32	AMUDHA	_	110/80	_	_	110/70	_	_	110/70	NIL	_	110/70	_			YES											
33	MALLIGA	_	120/80	_	_	120/80	_	_	120/80	NIL	_	140/100	NIL	YES												YES/LATE	
34	MARAGATHAM	_	110/80	_	_	110/70	_	_	120/80	NIL	_	120/70	_	YES													
35	SATHYAVANI	_	120/80	_	_	110/80	_	_	110/70	NIL	_	110/70	_	YES													
36	AMIRTHAVALLI	_	110/70	_	_	110/70	_	PE	110/70	NIL	PE	130/90	NIL		YES												
37	SARASWATHY		110/70			120/80		PE	130/80	_	_	150/90	NIL	_	_	_	_	YES	_	_	YES	_			YES	YES	
38	SHANMUGAM	_	100/70	_	_	120/70	_	_	120/70_		_	110/70	NIL	YES													
39	JEYAPRADHA	_	100/60	_	_	120/70	_	_	130/80	_	PE	140/90	PLUS		YES												
40	MARIYAMMAL	_	110/70	_	PT	110/70	_	PE	140/90	NIL	PE	140/90	NIL			YES											
41	ABIRAMI	_	20/60	_	_	100/60	_	_	120/80	_	_	120/80	NIL			YES										YES	
42	KOLANJIYAM	_	110/70	_	_	110/70	_	_	130/80	_	_	120/70	PLUS		YES												
43	KOKILA	PE	120/70	PLUS	PE	120/70	_	PE	140/90	NIL	PE	150/90	NIL				YES				YES				YES	YES	
44	GOMATHY	_	110/70	_	_	110/70	_	_	120/80	_	_	120/80	NIL				YES										
45	SHANTHI	_	120/70	_	_	110/70	_	_	130/80	_	_	130/80	NIL		YES												
46	KUSHPOO	_	110/70	_	_	120/80	_	_	110/70	_	_	120/70	NIL	YES													

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		PE	BP	UA	PE	BP	U/A	PE	BP	U/A	PE	BP	U/A	SPO	I	El.lscs	EM	A	D	assisted	PE	EC	Help	IUGR	PRETERM	RDS	
47	JHANANI	_	120/70	_	_	110/70	_	PE	140/90	PLUS	PE	140/90	PLUS		YES												
48	JEYASHREE	_	110/70	_	_	120/80	_	PE	140/80	NIL	PE	140/90	NIL	YES													
49	DHIVYAPRIYA	_	120/70	_	_	120/70	_	PE	140/80	NIL	PE	150/90	NIL						YES		YES				YES	YES	
50	BABYNISHA	_	110/70	_	_	110/70	_	_	120/80	_	_	130/80	NIL				YES										
51	DURGALAKSHMI	_	120/70	_	_	120/80	_	_	130/80	_	_	120/70	NIL				YES									YES	
52	JEMIR FATHIMA	_	110/70	_	_	110/70	_	PE	140/90	PLUS	PE	140/90	NIL				YES										
53	BRISILA ROSELIN MAR	_	120/70	_	_	120/80	_	_	120/80	_	_	120/70	NIL							YES							
54	ALAGUPRIYA	_	110/70	_	_	140/90	NIL	PE	140/80	NIL	PE	150/90	PLUS				YES								YES		
55	AMBIKA	_	110/70	_	_	130/90	NIL	_	130/90	NIL	_	140/100	PLUS				YES										
56	KAVITHA	_	120/80	_	_	110/70	NIL	_	110/70	NIL	_	110/70	NIL	YES													
57	GOMATHY	_	120/80	_	_	120/80	NIL	_	120/70	NIL	_	120/70	NIL				YES										
58	SHANTHI	_	120/70	_	_	120/80	NIL	_PE	110/70	NIL	_	100/60	NIL	YES													
59	MANGALAM	_	130/90	PLUS	_	130/90	PLUS	PE	130/90	PLUS	PE	140/100	PLUS			YES	YES										YES
60	SHARMILA DEVI	PE	120/80	_	_	120/70	NIL	PE	110/70	NIL	PE	120/70	NIL			YES											YES
61	MALARVIZHI	_	110/70	_	_	130/90	NIL	_	130/90	NIL	PE	140/90	PLUS														
62	DURGADEVI	_	120/80	_	_	120/80	NIL	PE	110/70	NIL	_	110/70	NIL	YES													
63	ANBUSELVI	PE	120/80	_	_	110/70	NIL	PE	110/70	NIL	PE	110/70	NIL				YES										
64	KALAIARASI	_	110/70	_	_	140/90	NIL	_	140/90	NIL	PE	140/90	NIL		YES												
65	MAGAMAYEE	_	120/80	_	_	110/70	NIL	_	110/70	NIL	_	110/70	NIL		YES												
66	RADHIKA	_	110/70	_	_	120/80	NIL	_	120/70	NIL	_	110/70	NIL	YES											YES		YES
67	SULTAN NACHAIR	_	110/70	_	_	150/90	NIL	_	150/90	PLUS	PE	150/90	PLUS				YES										
68	MARY JENNIFER	_	110/70	_	_	110/70	NIL	_	110/70	NIL	_	110/70	NIL	YES		YES											
69	ASHA BABY	_	120/80	_	_	120/80	NIL	_	120/70	NIL	_	120/80	NIL												YES		

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		PE	BP	UA	PE	BP	U/A	PE	BP	U/A	PE	BP	U/A	SPO	I	El.lscs	EM	A	D	assisted	PE	EC	Help	IUGR	PRETERM	RDS	
70	PANCHAVARNAM	_	120/80	_	_	140/90	NIL	PE	140/90	NIL	PE	150/90	NIL		YES												
71	KANAGA	_	110/70	_	_	140/90	NIL	PE	140/90	NIL	PE	110/70	NIL		YES												
72	PRIYADARSHINI	_	110/70	_	_	150/90	NIL	PE	140/80	NIL	PE	150/80	PLUS		YES												YES
73	MANGAYARKARASI	_	120/70	_	_	130/70	NIL	_	120/70	_	_	120/70	_			YES											
74	ANITHA	_	130/70	_	_	120/70	NIL	PE	130/90	NIL	PE	130/90	NIL		YES										MILD IUGR		
75	SUNDARI	_	120/70	_	_	120/90	NIL	_	130/80	_	_	130/70	_	YES													
76	NAZRIN BANU	_	120/70	_	_	100/60	NIL	PE	130/90	NIL	PE	140/90	PLUS		YES												
77	MATILDA	_	130/70	_	_	110/70	NIL	_	120/80	_	_	120/70	_	YES													
78	AASAIVALLI	_	120/70	_	_	100/60	NIL	_	130/80	NIL	PE	130/90	NIL		YES												YES
79	AFREINSITHIHA	_	120/80	_	_	110/70	NIL	_	120/70	_	_	130/70	_			YES											
80	AARTHI	_	110/70	_	_	120/70	NIL	PE	140/80	NIL	PE	140/90	NIL				YES								MILD IUGR		
81	VELANKANNI	_	110/70	_	_	110/70	NIL	_	120/80	_	_	120/70	NIL	YES													
82	ABIRAMI	_	110/70	_	_	120/80	NIL	_	120/80	_	PE	130/90	NIL		YES												YES
83	KAMALA	_	110/70	_	_	120/80	NIL	_	110/70	_	_	110/70	_	YES													
84	BARAKATH NISHA	_	110/70	_	_	120/80	NIL	_	110/70	_	_	110/70	_	YES													
85	NIRMALA	_	110/70	_	_	120/80	NIL	_	120/80	_	PE	130/90	NIL		YES										YES		
86	RANJITHA	_	110/70	_	_	110/70	NIL	_	120/80	_	PE	110/70	NIL	YES													
87	PUNITHAVALLI	_	110/70	_	_	110/70	NIL	_	120/80	_	_	120/70	NIL				YES									YES	
88	THINGALAVAL	_	120/70	_	_	120/70	NIL	_	110/80	_	_	120/80	NIL				YES										
89	DHARSHINI	_	120/70	_	_	110/70	NIL	_	110/70	_	_	110/70	NIL														
90	DHANYA	_	110/60	_	_	120/70	NIL	_	120/80	_	_	120/70	NIL														
91	KURINJIPOO	_	100/60	_	_	120/70	NIL	_	120/80	_	PE	130/90	NIL			YES									YES		YES
92	TAMILARASI	_	110/70	_	_	120/70	NIL	_	110/70	_	_	110/70	NIL	YES			YES										

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		PE	BP	UA	PE	BP	U/A	PE	BP	U/A	PE	BP	U/A	SPO	I	El.lscs	EM	A	D	assisted	PE	EC	Help	IUGR	PRETERM	RDS	
93	RADHIKA	_	120/80	_	_	110/80	NIL	_	120/80	_	_	120/80	NIL	YES													
94	MUTHULAKSHMI	_	110/70	_	_	120/80	NIL	_	110/70	_	_	120/80	NIL	YES													
95	MAHALAKSHMI	_	120/80	_	_	110/70	NIL	_	120/80	_	_	110/80	NIL		YES												
96	RAJINISHREE	_	110/70	_	PE	110/70	NIL	PE	120/80	_	PE	130/90	NIL	YES			YES								YES		
97	JASMNE PRABHA	_	120/80	_	_	110/70	NIL	_	110/80	_	_	120/70	NIL		YES												
98	ACQUINES RANI	_	110/70	_	_	120/70	NIL	_	120/70	_	PE	130/90	NIL				YES										YES
99	ILAKKIYA	_	120/80	_	_	110/80	NIL	_	120/80	_	_	120/80	NIL				YES										
100	RAMYA	_	110/70	_	_	120/80	NIL	_	120/80	_	_	120/80	NIL	YES													